Chapter One: Introduction

Extraction of teeth is considered an essential part of the dental practice. Teeth are extracted for many reasons including; dental caries, which is considered the most common reason, periodontal disease, endodontic failure, trauma, orthodontic or prosthodontics reasons, and many other reasons (Peterson, 2003).

The removal of a tooth is a challenge to the dentist because profound anesthesia is needed if it is to be removed without pain for the patient. Local anesthesia must be profound to eliminate sensation from the pulp, periodontal ligament and buccolingual soft tissues. Therefore, it is essential to anesthetize the nerves supplying the tooth to be extracted along with the surrounding hard and soft tissues (Peterson, 2003).

Most patients who are about to undergo tooth extraction will be anxious. A few patients face this procedure calmly and even patients with no overt signs of anxiety are likely to have internal feelings of distress (Peterson, 2003). Therefore, current standards of care dictate an adequate level of pain control which starts with preoperative medications including anesthesia, and continues into the postoperative period (Woodward, 2008).

The control of intra- and postoperative pain presents an age-old challenge since dental patients have become increasingly less tolerant of a dentist who hurts them (Hawkins and Moore, 2002), and because fear of pain is a main reason a patient may be apprehensive about dental treatment (Meechan, 2002). Therefore, pain control is an important factor to reduce the fear and anxiety associated with dental procedures (Pozos et al, 2006).

Local anesthesia forms the backbone of pain control in dentistry and is essential for pain-free dentistry (Malamed et al, 2001). Local anesthetic agents are the most frequently

administered drugs in dentistry and represent the primary method of pain control for patients undergoing intraoral procedures (Yagiela, 2000). Many local anesthetic agents have been introduced since the introduction of cocaine in 1884 (Malamed et al, 2001), with a variety of agents that provide rapid onset with adequate duration of anesthesia (Hawkins and Moore, 2002), among which lidocaine is the most popular (Srinivasan and Kavitha, 2009). Despite the fact that available local anesthetics are safe and effective, research has continued to seek safer and more effective ones (Malamed et al, 2001).

Anxiety about dental injections is common, even among well-educated people who seek care regularly. Fear of dental injections leads many patients to avoid dental care. Unfortunately, local anesthesia which is the most common means of pain control in dentistry can itself elicit anxiety. In fact, palatal injections are rated as one of the most traumatic procedures in dentistry by patients and dentists as well (Milgrom et al, 1997), and even though a number of adjunctive techniques have been described to reduce the discomfort of the palatal injection, they have not gained universal acceptance and a palatal injection is still a painful experience to the patient (Badcock et al, 2007).

Although they are painful and poorly tolerated by patients, palatal injections are still needed, as dictated by the anatomical description of the palatal sensory innervations, to anesthetize palatal soft tissues whenever any procedure that may involve manipulation of these tissues is performed. Therefore, the idea of anesthetizing the palatal mucosa without actually administering the painful palatal injection has emerged and the recent suggestion of the unnecessity of palatal injections for the extraction of maxillary teeth has gained an increasing interest.

Articaine hydrochloride is a relatively new amide local anesthetic agent that is believed to have superior anesthetic and better diffusion properties through hard and soft tissues than other local anesthetic agents (Paxton, 2008). Therefore, some authors have investigated the

possibility of extraction of maxillary teeth without a palatal injection by administering articaine buccal infiltration only and relying on its superior diffusion properties to anesthetize the palatal mucosa without the need for a separate palatal injection (Fan et al, 2009, Uckan et al, 2006).

1.1 Anatomy and Neurophysiology of The Oral Cavity

1.1.1 Nerve Supply of The Upper Jaw

The maxillary teeth, their supporting structures including the alveolar bone and the periodontal ligaments, and the mucosa covering the alveolar process are supplied by branches of the maxillary division of the trigeminal nerve, namely the posterior, middle and anterior superior alveolar nerves (Johnson and Moore, 1997).

The posterior superior alveolar nerve originates from the pterygopalatine ganglion. These branches pass through the pterygomaxillary fissure into the infratemporal fossa (Johnson and Moore, 1997), where they enter the maxilla through the posterior alveolar foramina at various levels. These nerves then pass antero-inferiorly across the posterolateral wall and then the floor of the sinus (Heasman, 1984), with some branches lying between the bone and the sinus membrane (Mc Daniel, 1956) to supply the maxillary molar teeth, the mucosa on the buccal surface of the associated alveolar process and the lining of the sinus (Johnson and Moore, 1997). Posterior superior alveolar nerve sometimes communicates directly with the descending anterior superior alveolar nerve (Heasman, 1984).

The anterior superior alveolar nerve originates from the infraorbital nerve as it passes the infraorbital canal. It descends in the sinuous canal downward close to the anterior wall of the maxillary sinus and the lower margin of the nasal aperture, to reach the incisors. It gives a small nasal branch to the mucosa of the lateral wall of the nasal cavity (Johnson and Moore, 1997). This nerve usually gives branches that communicate with the terminal

branches of the posterior and middle superior alveolar nerves (Heasman, 1984). It supplies the canine and the incisor regions (Johnson and Moore, 1997).

The middle superior alveolar nerve is not always present, but usually arises as a branch of the infraorbital nerve, anywhere between the inferior orbital fissure and the anterior superior alveolar branch, and passes anterio-inferiorly beneath the mucosa lining the posterior, lateral or anterior walls of the maxillary sinus (Johnson and Moore, 1997). Some variations in the origin of the middle superior alveolar nerve were found in 28% of cases (Fitzgerald, 1956), such as arising directly from the maxillary nerve in the pterygopalatine fossa or it may arise as a branch of the anterior superior alveolar nerve. It supplies the premolar teeth, and supplies fibers to the first molar (Johnson and Moore, 1997). It was found to be absent in 18% of cases, where its area is supplied by the overlapping of the adjacent nerves (Fitzgerald, 1956).

The superior alveolar nerve plexus is formed by the numerous branches of the superior alveolar nerves, and is located within the alveolar process above the apices of the roots of the maxillary teeth (Johnson and Moore, 1997), or in the bony floor of the sinus (Heasman, 1984). Branches of the plexus supply the maxillary teeth, the bone of the alveolar process, the periodontal ligaments and the mucosa on the buccal side of the alveolar process (Johnson and Moore, 1997).

The palatal mucosa is innervated by the greater palatine nerve posteriorly and the nasopalatine nerve anteriorly. The greater palatine nerve originates from the inferior aspect of the pterygopalatine ganglion and descends through the greater palatine canal to emerge through the greater palatine foramen on the oral surface of the hard palate, then it passes forward in a groove in the submucosa of the intermediate zone, approximately midway between the palatal gingival margin and the midline of the palate, to supply the mucosa

and the associated glands of the palate from the region of the greater palatine foramen to the canine or sometimes the lateral incisor region (Johnson and Moore, 1997).

The nasopalatine nerve is a branch of the pterygopalatine ganglion that runs with the medial nasal branches to the nasal septum, then it turns to pass downwards and forwards on the vomer to reach the incisive fossa, through which it emerges to the oral surface of the hard palate. It supplies a small part of the nasal septum and the mucosa in the incisor region of the hard palate (Johnson and Moore, 1997).

1.1.2 Neurophysiology of The Oral Cavity

The oral cavity has a high ratio of sensory innervation, which is demonstrated by the size of the sensory division of the trigeminal nerve and its innervation area as compared to the sensory roots of the trunk nerves and their innervation areas, which means that the oral cavity is densely populated with sensory receptors reflecting a high degree of discriminatory acuity. Furthermore, the innervation of the oral cavity has a very large representation in the cerebrum compared to innervation of body peripheral areas (Brill et al, 1962).

1.1.2.1 Neurophysiology of The Oral Mucosa

The oral mucosa is richly endowed with sensory nerve terminations, which show a great variation in their morphology. However, they are not subdivided into distinct morphological groups, but rather consist of fine non-myelinated terminal fibers which end freely in the epithelium and of many organized terminations of variable complexity (Dixon, 1961).

Free nerve endings were observed in all parts of the oral mucosa, they originate from the subepithelial nerve plexus which lies in a plane parallel to the surface of the mucosa. The plexus is made up of nerve bundles of variable thickness, presenting an inter-digitating pattern of both myelinated and non-myelinated fibers. Free and coiled nerve endings are

connected to this plexus, and this connection varies with the complexity of the end formation, the size of the subepithelial papilla or the profuseness of its innervation (Dixon, 1962).

These endings terminate either intrapapillary close to the basal layer of the epithelium or between the basal cells as fine intraepithelial fibers (Dixon, 1961). The proximity of intrapapillary nerve endings to the basal layer of epithelium is probably important in the localization of sensation (Dixon, 1962).

The majority of intraepithelial fibers are confined to the basal layers or deeper proliferating layers of the epithelium and only a small proportion extend to the superficial squamous layer. These intraepithelial fibers are probably associated with pain and touch sensations. Their distribution among the deeper layers of epithelium is correspondent with the known oral threshold of touch and pain, and if most of these fibers reached the surface layers of the epithelium then we might expect a much higher sensitivity of the oral mucosa than is known to exist. However, the high incidence of nerve endings in the deeper layers could account for the relatively high sensitivity of the oral mucosa compared with other regions of the body. Sensory discrimination on the other hand in the oral mucosa may depend not only on the incidence and position of nerve endings, but also on their mode of origin from the underlying nervous plexus (Dixon, 1962).

Although organized nerve endings vary in structure and size, they have the same characteristics in all parts of oral mucosa. They are classified into simple, complex and compound (Dixon, 1961). Many of them have delicate capsules close to the basal layers of epithelium, with actual contact sometimes. The functional importance of this intimate association is that the stimuli from epithelium might be more readily transmitted to the nerve termination (Dixon, 1962).

The physiology of sensibility is based on two contrasting theories, the specificity theory; in which it is assumed that every modality of sensation is associated with a specific end organ or nervous pathway, and the pattern theory which suggests that the difference in the manner of stimulation and the patterns of nerve impulses reaching the brain from nonspecific end organs differentiate one type of sensation from another (Dixon, 1961).

1.1.2.2 Neurophysiology of The Periodontium

In addition to the mucosa, the nerve fibers also innervate the periodontium of teeth. Nerve fibers supplying the periodontal ligament enter it in the apical region, and some through the lateral foramina in the alveolar bone, with the apical part being the most heavily innervated. The periodontal ligament contains myelinated and unmyelinated axons (Hildebrand et al, 1995), which are mostly located near the alveolar wall (Mason and Holland, 1993).

Nerve fibers in the periodontal ligament terminate either as free nerve endings or as specialized terminals, resembling Ruffini endings. Both types function as rapidly adapting mechanoreceptors. The response of the periodontal receptors is directly related to the amount of force applied, and to the direction of force. Some receptors are bidirectionally sensitive, and thus respond to tension and compression as well (Hildebrand et al, 1995).

1.1.3 Physiology of Nerve Conduction

After stimulation of the nerve, the transmission of impulses along the nerve occurs as a wave of electrical depolarization which is called an action potential. It is mediated by changes in the permeability of the nerve membrane to sodium. At rest this membrane is relatively impermeable to sodium but permeable to potassium. In addition a Na^+/K^+ ATPase pump actively transfers three sodium ions out of the cell in exchange of two potassium ions into the cell. This results in a higher extracellular sodium concentration and high intracellular potassium concentration, and consequently an electrical potential

difference across the membrane where the inside of the cell is negatively charged (-70 to -90 mV) compared to the outside (Lagan and McLure, 2004).

The membrane also contains voltage-gated sodium channels that open or close in response to changes in potential difference across the membrane. These sodium channels cycle through four states: resting, activated, inactivated and deactivated. In the resting state the channels are closed, stimulation of the nerve causes a conformational change in these channels which become activated and porous allowing the influx of sodium ions along an electrochemical gradient. This influx raises the membrane potential, and if sufficient sodium channels open and the membrane potential rises to a threshold value of -60 mV, this triggers opening of many sodium channels, resulting in a more rapid influx of sodium ions and the membrane potential may reach +20 mV, and then the channels become inactive (Lagan and McLure, 2004).

The depolarization of an area of the axon results in a positive charge of its intracellular portion relative to adjacent areas, producing a flow of ions and consequently an electrical current. The change in potential in the adjacent areas of the membrane causes opening of sodium channels and further depolarization. Thus, a wave of depolarization flows along the nerve transmitting the original stimulus (Lagan and McLure, 2004).

This electrical depolarization lasts for 1-2 ms, after which the membrane potential restores to its polarized state by the leakage of potassium and the active pumping of sodium. It remains deactivated and refractory to further stimulation for a short period, this refractory period prevents retrograde flow of the impulse and inhibits rapid re-depolarization of an axon that was originally depolarized (Lagan and McLure, 2004).

1.2 Pain

Pain is a personal subjective experience that is influenced by the cultural background of the individual, the meaning of the situation, attention, and other psychological variables (Katz

and Melzack, 1999). It is a sensory and emotional experience that is often associated with anxiety and increased activity of the sympathetic nervous system (Schwartz, 2006). It also has an unpleasant affective quality; it disrupts behavior and thought, becomes overwhelming and demands immediate attention (Katz and Melzack, 1999). Current theories support a multidimensional framework of pain experience with at least six major aspects; physiologic, sensory, affective, cognitive, behavioral and sociocultural (McGuire, 1992).

Earlier studies on pain focused on the concept that it is a purely sensory experience, while in fact its motivational affective properties that drive the patient into activity aimed at stopping the pain as quickly as possible, can't be ignored. Melzack and Casey suggested three major psychological dimensions of pain which are; (1) sensory discriminative which includes intensity, quality and location, (2) motivational affective which includes anxiety, depression and mood state, and (3) cognitive evaluative. These dimensions are served by specialized systems in the brain; the sensory discriminative dimension is affected by the rapidly conducting spinal systems, the motivational and unpleasant affective dimension is served by the reticular and limbic structures that are affected by the slowly conducting spinal systems, and the cognitive evaluative dimension is served by the neocortical or higher CNS processes, which also control the activity of the discriminative and motivational systems (Katz and Melzack, 1999).

These systems interact with each other to provide perceptual information on the magnitude, location and spatiotemporal properties of the painful stimuli, motivational tendency towards fight or flight, and cognitive information based on the past experience and the outcomes of different response strategies. All these together affect the motor mechanisms responsible for the complex pattern of responses to pain (Katz and Melzack, 1999).

1.2.1 Pathophysiology of Pain

It is well known that a basic understanding of the physiology of pain is essential for its management and for selecting modalities and agents best suited to every patient (Woodward, 2008). Pain is the sensation that is evoked by potential or actual noxious stimuli or by tissue injury, and is a major symptom of many diseases (Schaible and Richter, 2004). The perception of a noxious stimulus as pain requires its transmission as an electrical event from the site of injury to the higher brain centers or “nociception” (Schwartz, 2006), and thus pain is a subjective result of nociception, which is the encoding and processing of noxious stimuli in the nervous system (Schaible and Richter, 2004).

A noxious stimulus activates nociceptive pain receptors or nociceptors in the peripheral nerve. Most of these nociceptors are polymodal responding to noxious mechanical, thermal and chemical stimuli (Schaible and Richter, 2004). If the stimulus is strong enough, an action potential is triggered and conducted by the primary afferent nerves or first-order neurons to the dorsal horn of the spinal cord via the dorsal root ganglia (Schwartz, 2006, Fletcher and Spera, 2002).

Two different types of nerve fibers are associated with pain transmission; A-delta and C-polymodal fibers. The A fibers have a larger diameter and a thin myelin sheath. They are faster conducting (6-30m/s) and are thought to account for the more localized sharp, stabbing and electrical-type pain. On the other hand, C fibers have a smaller diameter, are unmyelinated and are slower conducting (0.5-2m/s). They are thought to be associated with the poorly localized duller, achy and more lingering pain (Schwartz, 2006, Fletcher and Spera, 2002).

Primary afferent stimuli enter the spinal cord, which is composed of various laminar tracts of second-order neurons, at the dorsal root zone at multiple levels. These stimuli either activate interneurons that are part of segmental motor or vegetative reflex pathways or they

cross to the contralateral ascending spinothalamic tract reaching the third-order neurons in the thalamus for further processing and activate the thalamocortical system, in which the afferent information is relayed to the somatotopic areas of the cerebral cortex where the conscious pain sensation arises. It is at this supraspinal level where factors such as fear, anxiety, depression and learned behavior influence the pain perception (Fletcher and Spera, 2002, Schwartz, 2006, Treede et al, 1999). The impulses pass through the brain stem to supply inputs to the reticular formation, limbic system, thalamus and somatosensory cortex in the brain. Pain fibers from the oral cavity are carried in the trigeminal nerve to the trigeminal ganglion. They enter the brain stem in the pontine region and cross to the opposite side where they ascend as the anterior trigeminothalamic tract to the thalamus and somatosensory cortex (Schwartz, 2006).

Descending inhibitory pathways from the cerebral cortex synapse in the dorsal horn and modify the pain sensations (Mehlisch et al, 1999), which could explain the gate control theory of pain by Melzack and Wall, in which emotional behavior and other sensory modalities can affect pain. Large mechanofibers and smaller pain fibers stimulate a target neuron in the dorsal horn, and thus when the large mechanofibers are stimulated pain perception is reduced. The individual's response to a noxious stimulus is determined by the response of the limbic system in addition to the emotional nature of pain. This may explain why patients respond in various ways to the same pain or why the same patient may respond differently to the same pain at different times (Schwartz, 2006).

When tissue cells are damaged, chemical mediators are released that stimulate nociceptors. These mediators include histamine, potassium chloride, polypeptides and serotonin (Schwartz, 2006). Nociceoptors can also release neuropeptides such as substance P and calcitonin gene-related peptide and thus induce inflammation with vasodilation, plasma

extravasation and attraction of macrophages. They also play a role in the modulation of transmitted afferent nerve stimuli (Fletcher and Spera, 2002, Schaible and Richter, 2004).

Other chemical mediators such as prostaglandins, which are synthesized and released during inflammation, sensitize nociceptors by lowering their activation threshold so that even light innocuous stimuli cause pain, and noxious stimuli evoke stronger responses than in the non-senstized nociceptors. In addition, silent nociceptors are recruited. These are C fibers that are inexcitable by noxious stimuli in normal tissues, but during inflammation they become sensitized and activated (Schaible and Richter, 2004).

1.2.2 Measurement of Pain

A scientific foundation of the studying of pain in patients requires the ability to assess and measure this pain (Katz and Melzack, 1999); to quantify its extent and degree, and provide a bias-free comparison of outcomes between patients (McGuire, 1992). In clinical practice, pain measurement is crucial to clinical diagnosis, treatment selection, evaluation of patients' response to intervention (Chapman et al, 1985), assessment of affectivity of new drugs (Katz and Melzack, 1999), and represents an overall appraisal and judgment of the patient's experience to guide clinical decision making. Therefore, the use of reliable and valid methods to aid the patient communicate the level of experienced pain is an essential part of good quality care and research (Briggs and Closs, 1999).

The search for valid methods for the evaluation of pain has revealed three strands of scientific effort; the first is psychophysics which measures the effect of analgesia by quantifying the noxious stimulation that elicits pain and the maximum tolerated stimulation, the second uses standardized questionnaires to categorize pain according to its emotional impact, distribution, character and other dimensions, and the third uses rating scales to measure pain intensity and is mainly used in clinical trials. Since the last one is self-reporting of pain by the patient, it is considered the crucial method by which analgesic

therapies are evaluated and compared (Noble et al, 2005). The visual analogue scale and the McGill pain questionnaire are probably the most frequently used self-rating measures for the measurement of pain in clinical and research settings (Katz and Melzack, 1999).

1.2.2.1 Visual Analogue Scale

The visual analogue scale is one of the most commonly used self-reporting measures of pain (Butler, 1997). It tries to measure a characteristic that is believed to range across a continuum of values which is not easy to measure directly. Pain for example, ranges across a continuum from none to an extreme amount, and from the patients perspective it appears as a continuous spectrum and does not take discrete jumps or categories. To capture this idea of a continuum the visual analogue scale was innovated (Crichton, 2001).

The millimeter scale is the most commonly used measurement interval, and with a 10 cm line, produces a possible range of scores from 0 to 100 (Butler, 1997). It can be a horizontal or a vertical line with two endpoints labeled “no pain” and “worst pain ever” or similar verbal descriptors (Katz and Melzack, 1999). It can be ungraduated or with obvious graduations which seems to be preferred by patients (Butler, 1997).

Patients are asked to place a mark on this line that represents the level of pain intensity they feel (Katz and Melzack, 1999). This requires the patient to be able to equate the length of line from its left end to the point marked with the amount of the experienced pain (Briggs and Closs, 1999). The distance from the low end of the scale to the patient’s mark is used as a numerical index of the severity of pain (Katz and Melzack, 1999).

The visual analogue scale is a unidimensional scale which measures only the sensory component or the intensity of the pain experience (Briggs and Closs, 1999). It can also be used to assess the amount of pain relief after administration of a treatment designed to reduce pain by asking the patients to rate the amount of pain at different points in time (Carlsson, 1983). This scale is sensitive to pharmacologic and nonpharmacologic

procedures that alter the pain experience (Choinière et al, 1990). It is more sensitive to changes in pain intensity than verbal scales because of the unlimited continuum of the visual analogue scale in contrast to the limited number of verbal descriptors in verbal scales (Ponce de Leon et al, 2004, Seymour, 1982). However, it may not produce reliable ratings with different groups of patients because each patient may interpret the scale differently (Briggs and Closs, 1999). It also highly correlates with measured pain on verbal and numerical rating scales (Briggs and Closs, 1999, Katz and Melzack, 1999).

Visual analogue scales are the measurement instrument of choice when a unidimensional measurement of pain is required (Katz and Melzack, 1999). Their advantages include their ease of administration and scoring (Jensen et al, 1986), rapid completion with minimal demands on ill patients (Briggs and Closs, 1999), reliability and validity (Briggs and Closs, 1999, Price et al, 1983, Seymour, 1982), suitability for repeated use (Butler, 1997), minimal intrusiveness and simplicity when clear instructions are given to the patients (Chapman et al, 1985, Katz and Melzack, 1999). Another major advantage of the visual analogue scale is its ratio scale properties. Ratio scale properties are essential in comparing levels of pain among different groups of patients and comparing different levels of pain within the same individual. In contrast to other pain measurement tools, equality of ratios are implied and thus validating assessment of percentage differences between measurements obtained at multiple points in time or from independent samples of subjects and enable both valid comparison between different types of pain and valid interpretations of analgesic efficacy (Katz and Melzack, 1999, Price et al, 1983).

On the other hand, several disadvantages and limitations of the standard visual analogue scale exist. These include its complexity since it requires the ability to translate a sensory experience into a linear form (Briggs and Closs, 1999), and not all patients understand the concept underlying the visual analogue scale as a graphic representation of pain severity.

Moreover, with the visual analogue scale it is more difficult to appreciate the clinical significance of a small change in patient's rating since it involves a small difference in position along an unmarked line rather than a different number as in the numeric rating scale (Marquiè et al, 2008), because the visual analogue scales may provide insufficient reference points for subjects either to recall previous responses or to record intensity changes accurately (Linton and Götestam, 1983).

In addition, it is difficult to administer in patients with organic brain syndromes, young children, heavily medicated patients, patients with multisystem diseases (Butler, 1997), those with perceptual-motor problems and patients who can't comprehend the instructions. It also has an impractical scoring method in a clinical setting in which immediate measurement of patients' responses may not be possible (Katz and Melzack, 1999).

The major disadvantage of the visual analogue scales is the supposition that pain is a unidimensional experience that can be measured with a single item scale. Although intensity is a prominent dimension of pain, pain actually represents an endless variety of unique qualities, rather than a specific single sensation that varies only in intensity or effect (Melzack, 1975, Katz and Melzack, 1999).

A lack of understanding of the visual analogue scale by the patient or poor instructions from the researcher may result in problems that seem more prevalent in the elderly patients. Another source of error is the use of the photocopier to reproduce the visual analogue scale since some photocopiers tend to slightly enlarge an image each time they provide a copy, resulting in a visual analogue scale that is longer than 10 cm (Briggs and Closs, 1999).

Furthermore, patient's rating of experienced pain was found to be highly sensitive to alterations in the end-point descriptors used to specify maximum pain intensity (Seymour,

1985). Also the patient's visual perspective and physical orientation to the scale, particularly the viewing angle may represent a source of error variance (Butler, 1997).

1.2.2.2 Verbal Rating Scales

Verbal rating scales consist of a series of verbal pain descriptors describing different levels of pain intensity and ordered from least to most intense, from which patients choose the word that best describes the intensity of their pain (Katz and Melzack, 1999, Briggs and Closs, 1999). They range from simple 4-point scales (from none to severe) (Seymour, 1982) to 15-point ratio scales (Gracely et al, 1978).

The main advantages of these scales include that they are simple to administer and have proved validity and reliability (Katz and Melzack, 1999), easy to score and have high compliance rates, rapid completion, can be read out by the interviewer (Briggs and Closs, 1999), are preferred by the elderly who find them easier to use (Kremer et al, 1981, Briggs and Closs, 1999), and with adequate number of categories have discriminative capacities that exceed those of unidimensional visual analogue scales (Jensen et al, 1994).

On the other hand, the major disadvantage of the verbal rating scales is their psychometric properties (Briggs and Closs, 1999), which means that because they separate the pain experience into distinct categories, they provide simple ranked data and the intervals between each category and the next can't be assumed to be equal (Heft and Parker, 1984). Consequently, the data they provide are ordinal, which limits the statistical analysis to nonparametric methods (Briggs and Closs, 1999).

Further disadvantages of the verbal rating scales are that they depend on patient's ability to read and understand the words, which is difficult for patients with limited vocabulary. Patients are also required to select from a limited number of descriptors and may not find a word that accurately represents their experience, or they may feel that they lie between two categories. These scales are also less sensitive to treatment effects than visual analogue

scales. However, sensitivity to change depends on the number of verbal descriptors used, and it seems that a scale with more than 11 items is likely to be as sensitive as a visual analogue scale to treatment effects (Briggs and Closs, 1999).

1.2.2.3 Numeric Rating and Verbally Administered Numeric Rating Scales

Numeric rating scales consist of a series of numbers ranging from 0 to 10 or 0 to 100, with the endpoints representing the extremes of the possible pain experience and labeled "no pain" and "worst possible pain" respectively, and patients choose the number that represents the intensity of their pain. The numeric rating scales are simple to administer, and have well-established validity and reliability (Katz and Melzack, 1999).

For the verbally administered numeric rating scale, patients are asked to indicate the intensity of pain by reporting a number that best represents it, usually between 0 and 10. It is easy and quick to administer verbally, and it has a demonstrated reliability and validity (Jensen et al, 1986, Marquiè et al, 2008, Morrison et al, 1998).

The verbally administered numeric rating scale scores were found to be highly correlated with the visual analogue scale scores in different pain states (Bijur et al, 2003, Holdgate et al, 2003, Marquiè et al, 2008), although it seems that the verbally administered numeric rating scale usually yields higher scores than the visual analogue scale. It is also easier for patients to understand and faster to administer than the visual analogue scale, whereas the visual analogue scale is better in translating changes in pain, and consequently evaluating the effect of treatment (Marquiè et al, 2008).

1.2.2.4 McGill Pain Questionnaire

In order to overcome the shortcomings of the aforementioned scales, Melzack and Torgerson (1971) developed a questionnaire to specify the qualities of pain. A total of 78 words that describe different aspects of pain were categorized into three major classes and 16 subclasses. The first class included words that describe the sensory qualities of pain in

terms of temporal, spatial, thermal, pressure and other properties and is comprised of 10 subclasses. The second class included words that describe the affective qualities in terms of tension, fear and autonomic properties that are part of pain and is comprised of 5 subclasses. Evaluative words comprised the third class which describes the subjective and overall intensity of pain, and includes only one subclass. Another 4 subclasses were added that included miscellaneous words that were rarely used but considered essential for the description of some types of pain, which makes a total of 20 subclasses (Melzack and Torgerson, 1971). This questionnaire which is known as the McGill Pain Questionnaire (Melzack, 1975) has become a widely used clinical and research tool (Katz and Melzack, 1999).

The advantages of this questionnaire include that it is valid, reliable, consistent and useful (Chapman et al, 1985, Katz and Melzack, 1999). It also provides a rapid measure of subjective pain (Melzack, 1975), and an insight into the qualities of experienced pain (Katz and Melzack, 1999). It is also sensitive to interventions aimed to reduce pain (Eija et al, 1995, Katz and Melzack, 1999, Nikolajsen et al, 1996), and it seems to provide a more sensitive measurement of mild postoperative pain than does a simple visual analogue scale because patients can be more precise in describing their pain by selecting appropriate descriptors (Katz et al, 1994).

One of the most prominent advantages of this questionnaire is its discriminative capacity, which aids in the differential diagnosis between various pain syndromes (Katz and Melzack, 1999). It has been shown that specific verbal descriptors of the questionnaire can discriminate between reversible and irreversible damage of nerve fibers of teeth (Grushka and Sessle, 1984). However, this discriminative capacity can be obscured by high levels of anxiety and certain keywords that discriminate specific syndromes may be absent (Katz and Melzack, 1999).

A major disadvantage of this questionnaire is that its successful completion relies on good verbal skills, which can create a strong cultural and educational bias. It is also considered by some researchers to be too long and demands sustained concentration by the pain sufferer (Walsh, 1984).

1.3 Local Anesthesia

Local anesthesia is defined as the loss of sensation in a circumscribed area of the body caused by depression of excitation in nerve endings or inhibition of the conduction process in peripheral nerves, without inducing loss of consciousness (Malamed, 1997). Local anesthesia forms the backbone of pain control in dentistry, with the aim of providing patients with pain-free care (Malamed et al, 2001).

1.3.1 Mechanism of Action

Local anesthetic agents cause temporary block of nerve impulses, and thus produce insensitivity to painful stimuli (Lagan and McLure, 2004). They diffuse through the lipophilic nerve membrane in the neutral unionized form. The ionized active form, which is produced by the lower intracellular pH, reversibly binds with the D4-S6 part of α subunit of the sodium channel. Consequently, sodium influx is reduced and if a sufficient number of sodium channels are blocked, the threshold potential of -60mV will not be reached, which will stop the impulse conduction (Hollmann et al, 2001, Lagan and McLure, 2004, Olschewski et al, 1998, Xiong and Strichartz, 1998).

In addition to affecting the intracellular portion of the sodium channel, the unionized local anesthetic also disrupts the intra-membrane portion of the channel and causes disordered expansion of the membrane. This block of nerve conduction can also be increased by blockade of potassium channels, calcium channels and G-protein-coupled receptors

(Hollmann et al, 2001, Lagan and McLure, 2004, Olschewski et al, 1998, Xiong and Strichartz, 1998).

The local anesthetic binding sites on the channels can be revealed or obscured by the channels' conformational changes, and thus the affinity of the local anesthetic for the channel varies with the channel state. The affinity is highest when the sodium channel is open or inactive, and least when it is closed. In addition, the affinity also varies between individual local anesthetics. Lidocaine for example, binds and dissociates quickly from the channel, whereas bupivacaine binds rapidly but dissociates more slowly (Lagan and McLure, 2004, Vanhoutte et al, 1991).

1.4 Local Anesthetic Agents

Local anesthetic agents are the most frequently administered drugs in dentistry, which represents the dentist's primary means of pain control. The availability of effective and safe local anesthetics has improved dental care and permitted the development of many surgical procedures available now to dental patients (Moore, 1984). The improvements in local anesthetic agents and techniques are probably the most important advances in dental science that have occurred in the past 100 years (Colombini et al, 2006).

Clinically useful local anesthetics are classified into ester or amide groups which differ in their chemical stability, pathway of metabolism and allergenicity (Lagan and McLure, 2004). Amide local anesthetics include lidocaine, mepivacaine, prilocaine, bupivacaine, etidocaine and articaine (Malamed, 1997).

1.4.1 Lidocaine

In 1943, a Swedish chemist, Nils Löfgren synthesized a new amide local anesthetic derived from xylidine and named it lidocaine, which was less allergenic and twice as potent as procaine. This new local anesthetic produced greater depth of anesthesia with a longer

duration over a larger area than a similar volume of procaine (Budenz, 2003). Lidocaine, which is also called lignocaine and Xylocaine, was introduced in 1947 (Lagan and McLure, 2004). Its proven efficacy combined with low allerginicity and toxicity over long term research and clinical use have confirmed its safety (Paxton, 2008). Therefore, it has become the prototypic dental local anesthetic in North America (Hawkins and Moore, 2002), replacing procaine as the drug of choice for pain control (Malamed, 1997). In addition to its anesthetic properties it is used intravenously as a class 1b anti-arrhythmic agent (Lagan and McLure, 2004).

Lidocaine is a tertiary amide derivative of diethylamino-acetic acid (Figure 1.1). It has a low pK_a (about 7.9) and thus it produces a profound block with a rapid onset in concentrations of 0.5-4%. Its protein binding is relatively low and so its duration of action is relatively short (Lagan and McLure, 2004).

Figure 1.1: Chemical Structure of Lidocaine

Lidocaine is metabolized in the liver by cytochrome P450 isoenzymes, resulting in N-hydroxylation and N-dealkylation of the drug (Simon et al, 1998). It is excreted via the kidneys, with less than 10% is unchanged and more than 80% as various metabolites (Malamed, 1997).

Lidocaine is formulated in cartridges as 2% lidocaine with 1:50000 epinephrine, 2% lidocaine with 1:100000 epinephrine, 2% lidocaine with 1:200000 epinephrine and 2% lidocaine without epinephrine (Hawkins and Moore, 2002). The 2% lidocaine with 1:50000

epinephrine is recommended only for hemostasis (Malamed, 1997), whereas the 2% lidocaine with 1:100000 epinephrine is the most commonly used anesthetic in the United States (Yagiela, 2000), and is considered the gold standard against which all new anesthetic agents are compared. Lidocaine 2% with 1:1000000 epinephrine rapidly induces oral anesthesia that lasts 90 to 180 minutes (Hawkins and Moore, 2002), with an onset of action from 2 to 5 minutes for maxillary infiltrations (Gross et al, 2007), an average pulpal anesthesia of 60 minutes in maxillary infiltrations and 85 minutes in mandibular block, 170 minutes of soft tissue anesthesia in maxillary infiltrations and 190 minutes for mandibular block (Ram and Amir, 2006). It was also found to provide the longest anesthesia of maxillary teeth after supraperiosteal injection (Danielsson et al, 1985). It was found that lidocaine had a higher success rate of infiltration anesthesia than bupivacaine (Gross et al, 2007), and was found to provide similar postoperative pain control and analgesic intake but better anesthesia and less hemorrhage than the long acting agents (Meechan and Blair, 1993).

The maximum recommended dose of lidocaine with or without epinephrine is 4.4 mg/kg of body weight, this dose allows a significant volume to be used to achieve profound anesthesia but with a reduced risk of development of toxic reactions (Malamed, 1997).

Lidocaine is also found in eutectic mixture of local anesthetics (EMLA). This oil/water emulsion contains 2.5% lidocaine and 2.5% prilocaine. It is a cream at room temperature and is able to penetrate skin easily; therefore it is used in pediatrics to reduce the pain of venepuncture (Lagan and McLure, 2004).

1.4.2 Articaine

Articaine was the first such drug to be investigated in dentistry in 1973 (Vree and Gielen, 2005). Although first prepared by Rusching and his colleagues in 1969 in Germany, carticaine was not used clinically until 1974. It was introduced in Germany in 1976 after

changing it generic name into articaine (Malamed et al, 2000, Lagan and McLure, 2004), and in Canada in 1986 where it became the most commonly used local anesthetic in dentistry, replacing lidocaine. Some 10 years later articaine was approved for use in dentistry in several European countries including the United Kingdom (Malamed, 2000), and was introduced in the United States in 2000 (Malamed et al, 2001). Articaine was reported to be the most widely used local anesthetic agent in dentistry in a number of countries including Germany, Italy, The Netherlands and Ontario, Canada (Gouws et al, 2004, Haas and Lennon, 1995).

Articaine is considered to be the drug of choice in dentistry in the vast majority of recent literature. It is widely used in dentistry for infiltration and block anesthesia because of the very fast onset and excellent quality of anesthesia, low degree of toxicity and short duration of action (Vree and Gielen, 2005).

The aforementioned advantages are based on the belief that articaine diffuses better through soft tissues and bone than other local anesthetics (Oertel et al, 1997). Articaine reversibly blocks nerve conduction through a mechanism similar to other amide local anesthetics. Epinephrine is included to delay the absorption of articaine thus prolonging the duration of anesthesia (Malamed et al, 2001).

Dental procedures that require local anesthesia are performed for the elderly and children, which may impose certain implications. Articaine was studied in the elderly subjects and it was found that its metabolism was age-independent since the activity of plasma esterases is age-independent (Oertel et al, 1999). This is a main difference from other amide local anesthetics which are metabolized in the liver, where the capacity of enzymes decreases with age (Sotaniemi et al, 1996). Therefore, changing the articaine dose for the elderly patients is not recommended nor needed (Oertel et al, 1999).

Articaine 4% was found to have a faster onset time in children compared to adults (Ram and Amir, 2006). It was also found that its serum concentration in children was comparable to those in adults, and it seemed that there was no need to recommend a lower mg/kg articaine dose limit for children because of age related differences in pharmacokinetics. In fact the relatively low maximum concentration and the short half life of 2% articaine are advantageous in pediatric dentistry (Jakobs et al, 1995).

1.4.2.1 Structure

The amide structure of articaine is similar to that of other local anesthetic agents. However, it is unique since unlike other amide local anesthetics it contains a thiophene ring, which contains sulfur, instead of benzene ring in its molecule and it also contains an additional ester group (Lagan and McLure, 2004, Oertel et al, 1997). The chemical formula of articaine hydrochloride is 4-methyl-3-[2-(propylamino) propionamido]-2-thiophene-carboxylic acid, methyl ester hydrochloride (Malamed et al, 2001), which belongs to the anilide group (Figure 1.2). The molecular weight of articaine is 320.84 g/mol, and the pure compound is water soluble in concentrations up to 20% (Winther and Patinuparusara, 1974, Ram and Amir, 2006).

Figure 1.2: Chemical structure of Articaine

Articaine is an analogue of prilocaine in which the benzene ring is replaced with the thiophene ring (Budenz, 2003). The thiophene ring increases the liposolubility of articaine,

and the ester group enables it to undergo biotransformation in the plasma, where it is hydrolyzed by plasma esterase, as well as in the liver by hepatic microsomal enzymes (Malamed et al, 2001).

1.4.2.2 Pharmacokinetics

All amide local anesthetics are metabolized in the liver (Oertel et al, 1999). However, the biotransformation of articaine occurs in both the plasma by pseudocholinesterases and in the liver by hepatic microsomal enzymes. Therefore, its plasma levels fall quickly (Lagan and McLure, 2004). In fact, it was found that 90-95% of articaine is metabolized in the blood, whereas only 5-10% is metabolized in the liver (Srinivasan and Kavitha, 2009).

Articaine has a very short plasma half life of approximately 27 minutes (Hawkins and Moore, 2002), which indicates a very low systemic toxicity, and consequently the possibility of repeated injections if needed. This along with similar analgesic efficacy allows the use of articaine in higher concentrations than other amide local anesthetics (Oertel et al, 1997). The main metabolic pathway of articaine is via hydrolysis of the carboxylic acid ester groups by serum esterases to give free carboxylic acid (Malamed et al, 2000), whereas the large carbomethoxy group prevents the hydrolysis of the amide bond (Simon et al, 1998). Articainic acid is the primary metabolite (Van Oss et al, 1989), of which 75% is excreted as such and 25% is glucuronidated (Vree and Gielen, 2005) by the tubular cells and excreted by the kidneys (Vree et al, 1997). However, additional inactive metabolites have been detected in animal studies (Malamed et al, 2000).

Studying the concentration dependence of articaine hydrolysis to articainic acid showed that saturable articaine metabolism was indicated by a higher articaine/articainic acid metabolic ratio, with higher articaine concentration in dental alveolar blood after dental extraction. The advantage of local saturation of the serum esterases is a desirable wide

toxic therapeutic ratio, and thus the persistence of the local anesthetic effect with low systemic toxicity (Oertel et al, 1996).

Plasma protein binding of articaine in patients varies between 50% and 70%, and that of articainic acid between 60% and 90%. Excretion of articaine is mainly by kidneys, with 2-5% excreted unchanged, 40-70% excreted as articainic acid and 4-15% excreted as its glucuronide. The percentage of the total dose recovered in the urine was between 50 and 91% and up to 100% in some studies (Van Oss et al, 1989, Vree and Gielen, 2005). However, the volumes of distribution and the total body clearance of articaine were about 10 times higher than its metabolite (Vree et al, 1997).

Renal clearance of articaine varies between 12 and 28 ml/min, while that of articainic acid is between 84 and 160 ml/min (Van Oss et al, 1989). Therefore, after articaine hydrolysis into articainic acid it is excreted renally, mainly by glomerular filtration, and up to 25% by renal conjugation. Consequently, regardless of the route of administration, articaine in the blood is hydrolyzed, with a part of it is conjugated, and then excreted (Vree and Gielen, 2005).

1.4.2.3 Clinical Properties

Articaine is used clinically as a 4% solution with epinephrine 1:100000 or 1:200000 (Malamed et al, 2001). The ideal epinephrine concentration to be used with articaine for best and safest clinical performance is controversial. Some authors reported that the epinephrine concentration whether its 1:100000 or 1:200000 in articaine 4% did not affect the clinical efficacy in terms of intraoperative bleeding, hemodynamic parameters, anesthetic properties and duration (Costa et al, 2005, Santos et al, 2007, Tófoli et al, 2003). Moreover, the plasma concentrations of articaine were found to be identical for both, which means that the 1:200000 epinephrine concentration is as adequate as the 1:100000 concentration in delaying systemic absorption of articaine (Hersh et al, 2006).

On the other hand, other authors reported that the duration of the anesthetic effect was related to the vasoconstrictor concentration in the anesthetic solution (Knoll-Köhler and Förtsch, 1992), and that more short term cardiovascular effects were experienced with the 1:100000 epinephrine than with the 1:200000 epinephrine (Hersh et al, 2006). However, although in theory these cardiovascular effects can be avoided by pre-injection aspiration which prevents intravascular injection, false negative results of aspiration are common (Tófoli et al, 2003). Furthermore, many authors stated that the amount of epinephrine in the local anesthetic formulation had a summative effect with the plasma catecholamine levels, and although this would not induce hemodynamic changes in young and healthy individuals, the risk of complications increases in patients with cardiovascular diseases. This led to the recommendation of lower concentration of epinephrine in anesthetic solutions (Colombini et al, 2006, Naftalin and Yagiela, 2002).

Articaine 2% with 1:200000 epinephrine has recently became available for dental use, and when it was compared to the 4% with 1:200000 epinephrine formulation, it was found that the duration of anesthesia was shorter with the 2% formulation. Otherwise, the 4% solution did not prove any superior anesthetic effect, and therefore, articaine 2% with 1:200000 epinephrine can be administered for tooth extractions, especially in children and the elderly who may benefit from the shortened soft tissue anesthesia and avoiding of the risk of overdosing (Hintze and Paessler, 2006).

Articaine initially appeared a less attractive agent for dental procedures because of the higher per cartridge unit cost. However, its slightly faster onset of action, longer and more profound anesthesia with a greater ability to diffuse through tissues made it a widely used anesthetic in Europe and Canada (Budenz, 2003). The maximum recommended dose of articaine for adults is 7 mg/kg body weight, whereas the maximum recommended dose for children below 12 years of age is 5 mg/kg (Wright et al, 1989).

The onset of anesthesia for articaine 4% with epinephrine 1:200000 was found to be 1.5 to 1.8 minutes for maxillary infiltration and 1.4 to 3.6 minutes for inferior alveolar nerve block. Other studies have found that the onset of anesthesia is 1.5-3.0 minutes for maxillary infiltrations and only slightly longer for inferior alveolar blocks (Cowan, 1977, Malamed et al, 2001, Hawkins and Moore, 2002).

Duration of anesthesia with articaine was found to be sufficient for most dental procedures (Cowan, 1977). It was found to provide pulpal anesthesia of 60-75 minutes (Sherman et al, 2008). On the other hand, the duration of soft tissue anesthesia for maxillary infiltration varied between studies. Cowan (1977) reported an average duration of 2 hours 15 minutes ± 1 hour 20 minutes. The long duration of articaine is advantageous for permitting the completion of the dental procedure, particularly when long appointments are anticipated such as full mouth periodontal surgery or multiple implant placements (Hawkins and Moore, 2002).

The concentration of articaine in the alveolus of a tooth in the upper jaw after extraction was about 100 times higher than that in the systemic circulation (Oertel et al, 1997). This saturable local articaine metabolism possibly contributes to the long duration of local anesthetic effect despite its very short systemic half life (Vree and Gielen, 2005). The long anesthetic duration of articaine can also be explained by its higher protein binding which was found to be 95% as compared to that of lidocaine which was found to be only 65%. Local anesthetic agents are believed to act by binding to a protein receptor in the sodium channel, thus, the greater protein binding of an agent presumably results in a longer period of sodium channel blocking and a longer duration of anesthesia (Malamed et al, 2000, Ram and Amir, 2006). Furthermore, the long duration of postoperative anesthesia with articaine can be explained by its ability to diffuse through tissues (Vree and Gielen, 2005).

The basis of the wide use of articaine, although supported by little data, is the belief that articaine has superior diffusion properties and thus provides more effective anesthesia than other local anesthetics (Hawkins and Moore, 2002). These “superior diffusion” properties of articaine are founded on the notion that its thiophene ring increases its liposolubility and thereby its ability to penetrate nerve membranes (Srinivasan and Kavitha, 2009).

Many authors suggested that the claimed superior diffusion allows articaine to induce lingual and palatal anesthesia following buccal infiltration (Hawkins and Moore, 2002). Decades ago, Frankel suggested that the extent of anesthesia with articaine is so intense that lingual and palatal anesthesia could be obtained from buccal infiltrations in the premolar region (Cowan, 1977). It was also noticed that in a variable percentage of patients, adequate palatal anesthesia for tooth extraction can be obtained by a maxillary infiltration injection in the buccal vestibule. Similar results have been claimed for the mandibular anterior and premolar teeth with buccal infiltrations (Budenz, 2003).

On the other hand, other authors suggested that these superiority claims were based on speculations since the majority of literature failed to support them (Paxton, 2008). The ability of articaine to induce mandibular pulpal anesthesia and anesthesia of lingual or palatal tissues after labial administration could not be proved. Compared to prilocaine when used as labial infiltration for canines and second molars, no superior anesthetic efficacy of articaine was found for mandibular pulpal, lingual or maxillary palatal tissues (Haas et al, 1990). Oliveira et al (2004) also did not find any difference in palatal pain sensitivity after buccal infiltration between articaine and lidocaine, and thus once again the better diffusion of articaine could not be proved.

Despite the “gold standard” position of lidocaine, many reports have awarded articaine a superior reputation primarily based on the belief of its enhanced anesthetic properties (Paxton, 2008). However, because articaine is a comparatively new anesthetic agent, it is

still the subject of many discussions among dental surgeons (Tortamano et al, 2009). Many studies evaluated the clinical efficacy of articaine and even it compared it to other local anesthetics, of which lidocaine was the most eligible candidate.

Results of comparative studies with lidocaine have been contradictory, in part because of the different amounts and concentrations of anesthetic agents and epinephrine that were used, along with the use of different assessment methods of anesthesia, of which some authors may have used are pulp testers whereas others assessed lip numbness. However in solutions with equal concentrations of epinephrine, articaine was found to provide faster and more prolonged anesthesia than lidocaine (Vähätalo et al, 1993).

A meta-analysis by Paxton revealed that articaine was associated with increased anesthetic success in dental procedures (Paxton, 2008). Articaine was reported to have a potency 1.5 times and a toxicity of 0.6 times that of lidocaine, and a potency of 1.9 times and a toxicity of 0.8 times of procaine (Wright et al, 1989). Furthermore, an in vitro study showed that 2% and 4% articaine were more effective than 2% and 4% lidocaine, and 3% mepivacaine in depressing the action potential of the A fibers in rats (Potočnik et al, 2006). In addition, the maximum acceptable dosage for lidocaine in adults is 300 mg and for procaine is 400 mg, whereas for articaine it is 500 mg. Therefore, its greater potency and safety support its use in children, where minimum effective dose is recommended (Wright et al, 1989).

Winther and Nathalang conducted the first clinical trial to test the efficacy of articaine in Denmark in 1972, in which they found that 2% articaine was significantly superior to 2% lidocaine in terms of duration and extent of anesthesia (Paxton, 2008).

Kanaa et al (2006) and Robertson et al (2007) found that a buccal infiltration of 4% articaine with 1:100000 epinephrine was more effective than a similar injection of 2% lidocaine with 1:100000 epinephrine in producing pulpal anesthesia for mandibular molars. Whereas Srinivasan and Kavitha (2009) found that 4% articaine was superior to 2%

lidocaine in producing pulpal anesthesia in maxillary posterior teeth after buccal infiltration, and Costa et al (2005) found that articaine 4% with 1:100000 and 1:200000 epinephrine provided shorter onset and longer duration pulpal anesthesia in maxillary infiltration of posterior teeth than lidocaine.

This higher efficacy was believed to be either due to a greater diffusion of articaine (Oertel et al, 1997, Corbett et al, 2008) or simply the result of a concentration effect (Malamed et al, 2000). Furthermore, the success and efficacy of mandibular infiltration with articaine 4% for first molars was found to be comparable to that of inferior alveolar nerve block over 30 minutes (Jung et al, 2008), and thus providing an alternative to inferior alveolar nerve block in certain cases (Corbett et al, 2008).

Corbett et al (2008) found that the efficacy and the onset of pulpal anesthesia in mandibular first molar did not differ with buccal infiltration of articaine and epinephrine as compared to buccal and lingual infiltration. On the other hand for lidocaine, Meechan and Ledvinka (2002) found that buccal and lingual infiltration of lidocaine is more effective than buccal infiltration alone in obtaining pulpal anesthesia of lower anterior teeth.

Evans et al (2008) found that articaine 4% with 1:100000 epinephrine improved anesthetic success after maxillary infiltration in the lateral incisor region compared to lidocaine 2% with 1:100000 epinephrine. However, they had similar anesthetic success for first molars. Oliveira et al (2004) found that the anesthetic efficacy of articaine and lidocaine after buccal and palatal infiltration of maxillary canines was similar. However, articaine seemed to provide longer duration of pulpal anesthesia.

On the other hand, although many studies supported the superior anesthetic properties of articaine compared to lidocaine (Malamed et al, 2001), others failed to demonstrate the superior anesthetic efficacy of articaine over lidocaine in mandibular teeth, maxillary lateral incisors, canines and molars (Mikesell et al, 2005, Malamed et al, 2001, Claffey et

al, 2004, Sherman et al, 2008, Berlin et al, 2005, Malamed et al, 2000, Vähätalo et al, 1993, Oliveira et al, 2004, Evans et al, 2008). Thereby, the superiority of articaine over lidocaine could not be statistically verified by a clinical study (Tortamano et al, 2009).

Furthermore, several studies reported similar anesthetic efficacy between 4% articaine and 2% lidocaine when used for inferior alveolar nerve block (Claffey et al, 2004, Mikesell et al, 2005, Malamed et al, 2000, Evans et al, 2008), intraligamentary injection (Berlin et al, 2005), infiltration injection (Malamed et al 2001, Oliveira et al, 2004, Sherman et al, 2008, Srinivasan and Kavitha, 2009), Gow-Gates injection (Sherman et al, 2008) and supplemental buccal infiltration for inadequate pulpal anesthesia (Rosenberg et al, 2007).

Ram and Amir (2006) reported that articaine 4% with 1:200000 epinephrine was as effective as lidocaine 2% with 1:100000 epinephrine when used for children. They both had similar onset of anesthesia and efficacy but articaine had a significantly longer duration of soft tissue numbness. They also suggested that the similar efficacy of articaine 4% to lidocaine 2%, although they had different concentrations might be explained by the fact that lipid solubility of articaine was 1.5 while for lidocaine it was 4.0 (Ram and Amir, 2006), keeping in mind that lipid solubility is a determinant of potency of the anesthetic agent in vitro. However, in vivo higher lipid solubility may also cause redistribution of the drug to fat stores, and thus decreasing its potency (Schecter and Swisher, 2000).

It was found that postinjection pain after wearing of the anesthesia was higher with articaine than with lidocaine. Nevertheless, this pain was mild and clinically insignificant (Evans et al, 2008). Furthermore, both lidocaine and articaine have the same maximum dose of 500 mg (6.6 to 7 mg/kg) for the adult patient. However, because articaine is marketed as a 4% solution the maximum recommended dose for a healthy 70-kg adult is 7 cartridges of articaine compared to 13 cartridges of lidocaine 2% solution (Malamed, 1997).

1.4.2.4 Adverse Effects

Several studies reported that articaine was safe local anesthetic, with few reported problems of parasthesia, dysesthesia or prolonged anesthesia (Wright et al, 1989, Oertel et al, 1999, Malamed, 2000). The tolerance of articaine was found to be comparable to that of lidocaine (Cowan, 1977). The overall incidence of adverse events of articaine was also found to be similar to that of lidocaine. The most frequently reported adverse events after local anesthetic administration, excluding post-procedural dental pain, was headache (4%), facial oedema (1%), infection (1%), gingivitis (1%) and paresthesia and hypestheisa (1%). The incidence of these events was found to be similar between lidocaine and articaine. Whereas, the adverse events most frequently reported as related to articaine use were paresthesia (0.9%), hypaesthesia (0.7%), headache (0.55%), infection (0.45%), rash (0.3%) and pain (0.3%) (Malamed et al, 2001). Other authors reported edema of lips and eyelids, trismus, soreness and swelling (Colombini et al, 2006).

One of the most controversial aspects of articaine administration is its potential to cause parasthesia after inferior alveolar nerve block (Haas and Lennon, 1995), which is why some researchers recommended that articaine 4% should not be used for this anesthetic technique (Hillerup and Jensen, 2006). Although Malamed et al (2001) reported similar incidence of parasthesia with articaine and lidocaine, Haas and Lennon (1995) reported that articaine and prilocaine were associated with a slightly higher incidence of mandibular and lingual parasthesia compared to other local anesthetics. This might support the suggestion that local anesthetics might have the potential for mild neurotoxicity at higher concentrations (Haas and Lennon, 1995). It was thus suggested that the anesthetic dose should be reduced to the absolute minimum amount required for effective anesthesia along with a slow atraumatic injection technique that is preceded by repeated aspirations (Budenz, 2003). It may be possible to decrease the risk of parasthesia by using a lower

concentration of articaine such as the 2% formulation which was recently introduced in Germany, and was found to be as effective as the 4% formulation in tooth extraction with infiltration anesthesia (Hintze and Paessler, 2006).

Some authors reported postsurgical complications of localized osteitis, mucosal ulcerations and sharp pain in patients who received articaine 4% with epinephrine 1:100000. On the other hand, other investigators did not report any local reactions or secondary effects after 500 and 7500 injections with articaine 4% with 1:200000 epinephrine (Malamed et al, 2001).

Malamed et al (2001) did not observe any consistent changes in the vital signs after the injection of articaine or lidocaine or at the end of the dental procedure. However, transient increases and decreases in blood pressure, heart rate and respiratory rate were observed, which were not clinically significant, and might be attributed to the anxiety caused by the injection itself or the impending dental procedure (Malamed et al, 2001).

Articaine 4% with 1:200000 epinephrine can be used for dental anesthesia in most cardiovascular patients. In fact, Elad et al (2008) found that articaine was as safe as lidocaine 2% with 1:100000 epinephrine in cardiovascular patients, despite the differences of the concentration of the local anesthetic agent and vasoconstrictor. However, the procedures they performed for these patients were relatively nonstressful, which makes it difficult to generalize their conclusions about the safety of articaine to include more stressful procedures such as extractions (Elad et al, 2008). Thus, articaine should be used with caution in patients with significant impairments in cardiovascular function and hepatic disease, since amide anesthetics undergo biotransformation in the liver and they have myocardial depressant properties (Malamed et al, 2001).

Ophthalmologic complications after anesthetizing the posterior superior alveolar nerve may occur. The most common is paralysis of the oculomotor muscles causing diplopia.

These effects usually occur few minutes after the injection of the anesthetic, however they are transient and complete resolution on cessation of the anesthetic effect is noticed (Peñarrocha-Diago and Sanchis-Bielsa, 2000).

The pathologic mechanism is thought to be caused by diffusion of the anesthetic solution from the pterygomaxillary fossa to the orbital region, possibly through the vascular, lymphatic or nervous networks connecting the pterygoid venous plexus and the orbit, resulting in direct effect on the nerve endings in the orbit. A defect in the bony cavity of the maxillary sinus will facilitate such diffusion (Peñarrocha-Diago and Sanchis-Bielsa, 2000).

Although most cases of ophthalmologic complications in the literature were caused by both lidocaine and Mepivacaine, these complications can be caused by articaine either because of its increased bone and soft tissue diffusion or because it is likely to cause interruption of motor and sensory pathways (Peñarrocha-Diago and Sanchis-Bielsa, 2000).

The immunogenic potential of articaine is very low (Malamed et al, 2001), due in part to the lack of metabolites derived from the benzene ring (Santos et al, 2007). Allerginicity of articaine does not seem to be greater than other available dental anesthetic agents, which is extremely rare (Hawkins and Moore, 2002). However, sodium bisulfate, the antioxidant included in anesthetic solutions that contain epinephrine, may cause allergic reactions. Some articaine solutions may contain the antibacterial preservative methylparaben, which may have contributed to the allergic reactions that have been reported with articaine (Malamed et al, 2001).

Allergic reactions that were reported with articaine include edema, erythema, urticaria and anaphylactic shock. Malamed et al (2001) reported that rash or pruritis were not more frequent with articaine than with lidocaine, and no serious allergic reactions were seen with either. Furthermore, patients allergic to articaine would likely be allergic to lidocaine and

other amide local anesthetics. Therefore, articaine with epinephrine is contraindicated in patients with known sensitivity to amide local anesthetics and in patients with sulfite sensitivity, such as allergic-type asthma (Malamed et al, 2001).

Erythmatous, non allergic, non itching skin rashes were noted in 80% of cases with articaine and 20% of cases with prilocaine when used for intravenous regional anesthesia of the upper extremities. These rashes appeared only in the areas exposed to the anesthetic and disappeared within an hour. It was thought that these rashes might be a sign of venous endothelial irritation that were caused by direct toxic reaction in the small venules and capillaries (Pitkänen et al, 1999).

Reports of toxicity reactions are extremely rare, and the apparent lack of overdose reactions reported following articaine administration, even though it is marketed as a 4% solution, may be explained by its rapid inactivation by plasma esterases (Hawkins and Moore, 2002). However, although tissue toxicity is rare, there are few case reports of skin erythma, wheals and acute allergic reaction after mandibular block with articaine (MacColl and Young, 1989, Malanin and Kalimo, 1995).

Although the risk of systemic toxicity with articaine was reported to be low, its safe use in pregnancy and lactation is not yet established, and its use in children younger than 4 years is not recommended (Malamed et al, 2001), although Wright et al (1989) supported its use for this age group and did not report any adverse effects.

So far, Published data have supported the overall safety and tolerance of articaine with epinephrine. Therefore, articaine is considered a well tolerated and safe local anesthetic for use in dentistry, and can be used effectively in both adults and children, with a low risk of toxicity that seems comparable to that of other local anesthetics (Malamed et al, 2001, Wright et al, 1989)

Chapter Two: Aims and Objectives

2.1 Aims

The general aim of the study was to demonstrate whether adequate palatal anaesthesia for extraction of maxillary teeth can be provided by a buccal local anesthetic injection alone as compared to buccal and palatal injections.

2.2 Objectives

The objectives of the study included comparing the patients' perception of injection pain during buccal and palatal injections and to demonstrate whether articaine/HCL will provide superior anaesthesia in the palate as compared to lidocaine if both were administered as a single buccal injection for the extraction of permanent maxillary teeth.

Chapter Three: Literature Review

3.1 Extraction of Maxillary Teeth

Extraction of teeth in general is performed for a variety of reasons, and even though modern dentistry recommends that all possible measures should be taken to preserve the teeth in the oral cavity, it is still necessary to remove some of them. Severe caries that renders the tooth non restorable is probably the most common and widely accepted reason for tooth extraction. Many other reasons include; pulp necrosis or irreversible pulpitis that is not amenable to root canal treatment, endodontic failure, severe and extensive periodontal disease with irreversible tooth mobility, orthodontic reasons to provide space for tooth alignment, malpositioned teeth that traumatize oral soft tissues and cannot be repositioned by orthodontic treatment, malopposed teeth such as hypererupted teeth that interfere with prosthesis construction, painful cracked tooth and tooth with a fractured root that can’t be managed by a more conservative technique, teeth that interfere with the design and proper placement of prosthetic appliances, supernumerary teeth that is interfering with the eruption of succedaneous teeth, teeth associated with pathologic lesions, teeth in the line of radiation therapy, teeth involved in jaw fractures, esthetic reasons such as severe staining or malposition and economic reasons (Peterson, 2003).

Profound anesthesia is needed if the tooth is to be extracted without causing pain to the patient, and thus it is essential that surgeon knows the precise innervations of all teeth and the surrounding soft tissues in addition to the necessary injection techniques needed to anesthetize those nerves (Peterson, 2003).

3.1.1 Local Anesthetic Techniques of The Maxilla

The oral cavity receives most of its innervations from the maxillary and mandibular branches of the trigeminal nerve. These branches can be anesthetized by intraoral and extraoral techniques (Yagiela, 2000). Extraoral injections have been used for the relief of pain in trigeminal neuralgia, in cases of trismus and acute infections (Oringer, 1949). Even though effective, extraoral techniques are more painful, more invasive and more difficult than intraoral techniques. During the past century, specialized equipments and drugs were developed to facilitate intraoral injections (Yagiela, 2000).

Regional anesthetic techniques are used for many common oral procedures, including extractions, periodontal flap surgery, treatment of traumatic injuries of the oral cavity, tumor removal, palatal surgery, periodontal therapy, and root canal therapy (Woodward, 2008). Painless administration of anesthesia is as essential to the end result as is the effectiveness of the anesthetic agent. Relatively painless administration of the needle can be assured by following a few basic rules; the needle must be sharp and free of barbs and the tissues must be stretched gently so that the needle penetration of the surface layers can be quick and without pressure. A drop of anesthetic solution should be deposited immediately after initial penetration before the needle is forced deeper into the tissues. Slow rate of injection with the anesthetic solution used at body temperature will also reduce the pain of the injection. Topical anesthesia also helps to reduce initial pain from injections (Oringer, 1949).

Providing adequate anesthesia in the maxilla is rarely a problem, since the thin maxillary bone allows the ready diffusion of the local anesthetic to the apex of the tooth to be treated. Therefore, many dentists rely only on supraperiosteal anesthesia for most treatments in the maxilla (Malamed, 1997). However, despite the wide spread use of local anesthesia,

unsatisfactory anesthetic results might occur which is attributed to improper anesthetic technique rather than inherent deficiency of the anesthetic drug (Oringer, 1949).

Many factors may affect the success of local anesthesia, some are within the dentist's control and some are not. Therefore, guidelines that can help reduce the incidence of anesthetic failure exist. If the depth or duration of anesthesia after the first anesthetic injection was poor, a second injection into the same site might be adequate simply because of the increased volume of the anesthetic solution. However, using a different anesthetic agent for the second injection might increase its success, due to individual variances of tissue pH conditions along with different chemical properties of each anesthetic agent (Budenz, 2003).

Anesthetic failure is defined as inadequate depth and/or duration of anesthesia to begin or continue a dental procedure. This can be attributed to a variety of factors of which, assuming correct anesthetic technique, thicker cortical plates, denser trabecular pattern, variable innervation pathways or more myelin-rich nerve bundles. Inadequate anesthesia occur more common in the mandible than in the maxilla, nevertheless maxillary failures do occur and is frustrating as well (Budenz, 2003).

Most maxillary anesthesia failures, when exist, are attributed to individual anatomical variances of nerve pathways through the maxillary bone. Although maxillary pulpal sensory innervations are mainly propagated by the anterior, middle and posterior superior alveolar nerves which also supply the buccal soft tissues, accessory pulpal innervations may also be supplied from the greater palatine or nasopalatine nerves (Budenz, 2003).

The pulpally involved tooth is the most encountered difficulty, which is because of infection or inflammation the use of the supraperiosteal injection is contraindicated or ineffective. The most often problems in achieving adequate pulpal anesthesia via supraperiosteal injection in nonpulpally involved teeth is in the central incisor whose apex

may lie beneath the denser bone of the nose, the canine which may have a very long root with the anesthetic solution deposited below the apex, and the molars whose buccal root apices may be covered by the dense bone of the zygomatic arch and whose palatal root may flare toward the palate making the distance of the local anesthetic diffusion too great. However, in such situations the use of regional block anesthesia is essential to control pain (Malamed, 1997).

3.1.1.1 Supraperiosteal Injection

Supraperiosteal injection, more commonly but incorrectly called local infiltration, is the most frequently used local anesthetic technique for obtaining pulpal anesthesia in maxillary teeth (Malamed, 1997). It is of the most popular and universal methods of anesthesia for dental procedures for its simplicity and high degree of safety of administration. The maximum usefulness of infiltration is largely confined to the maxilla, although many nutrient foramina are present on the labial and lingual surfaces of the mandible through which effective infiltration anesthesia may sometimes be obtained (Oringer, 1949).

Supraperiosteal injection in the maxilla anesthetizes the large terminal branches of the dental plexus and results in anesthetizing the pulp and root area of the tooth, buccal periosteum, connective tissues and mucosa. It has many advantages of which is that it has a high success rate, and it is an easy and atraumatic injection. It is indicated for dental procedures that are confined to a relatively circumscribed area in either the maxilla or the mandible, such as pulpal anesthesia of the maxillary teeth when treatment is confined to one or two teeth or when soft tissue anesthesia is required for surgical procedures in a circumscribed area (Malamed, 1997). In addition, although some authors believe that it is contraindicated in the presence of infection, others suggested that infiltrations anterior and posterior to the area of localized infection should provide enough anesthesia (Yagiela, 2000).

However, when dental procedures are performed on a larger area or on multiple teeth, regional blocks are preferred since multiple supraperiosteal injections will be needed that require many needle penetrations each with the potential of causing pain either during the injection or after recovery from the anesthesia. Furthermore, using multiple supraperiosteal injections leads to the administration of a larger volume of local anesthetic solution with an increase in the risk of systemic and local complications (Malamed, 1997).

After lifting the lip and pulling the tissue tight, and with the syringe parallel to the long axis of the tooth, a 25- or 27- gauge needle is inserted at the height of the mucobuccal fold above the apex of the tooth to be anesthetized with the bevel of the needle directed toward bone, until the bevel is at or above the apical region of the tooth. In most cases the depth of penetration will be only a few millimeters, and since the needle is in soft tissues and is not touching the bone, no resistance should be felt to its advancement nor any patient discomfort during the injection (Malamed, 1997). Furthermore, supraperiosteal injections of the incisors should avoid the midline because of the highly sensitive periosteum of the anterior nasal spine (Yagiela, 2000). After negative aspiration, the anesthetic solution is deposited in a slow rate with the avoidance of tissue ballooning (Malamed, 1997), with the usual volume of the injected local anesthetic solution is 1 to 2 ml (Yagiela, 2000).

Failure of this injection, although rare, is either because the needle tip lies below the apex with the deposition of the anesthetic solution below the apex of the tooth leading to excellent soft tissue anesthesia and poor pulpal anesthesia, or the needle tip lies too far from the bone in which the solution is deposited in the buccal soft tissues (Malamed, 1997).

3.1.1.2 Greater Palatine Nerve Block

The greater palatine nerve block is indicated for dental procedures involving the palatal soft tissues distal to the canine such as restorative procedures on more than two teeth that

require subgingival manipulation and for pain control during periodontal or oral surgical procedures involving the palatal soft and hard tissues. Even though it is considered traumatic, the greater palatine nerve block is less traumatic than the nasopalatine nerve block because the tissues surrounding the greater palatine foramen are more able to accommodate the volume of the deposited solution. This block anesthetizes the posterior portion of the hard palate and its overlying mucoperiosteum as far anteriorly as the fist premolar and medially to the midline (Malamed, 1997).

The advantage of this block is that it minimizes needle penetrations and volume of injected anesthetic solution and thereby reducing patient discomfort. However, a major disadvantage of this technique is that it is potentially traumatic. After locating the greater palatine foramen which is most frequently located distal to the maxillary second molar, a 27-gauge short needle is advanced from the opposite side of the mouth at a right angle and is inserted in the palatal mucosa anterior to the greater palatine foramen with the bevel toward the palatal soft tissues. While depositing small volumes of the anesthetic, the needle is slowly advanced to the target area, which is the greater palatine nerve as it passes anteriorly between the mucosa and bone of the hard palate, until palatal bone is contacted. The depth of penetration at this point will be less than 10mm, and after negative aspiration the anesthetic solution is slowly deposited (Malamed, 1997).

Failure of this block is either due to deposition of the local anesthetic too far anterior to the foramen or inadequate anesthesia opposite the first premolar caused by overlapping fibers from the nasopalatine nerve. Complications of this technique include; ischemia and necrosis of palatal mucosa when high concentrations of vasoconstrictor is used for hemostasis over a prolonged period, the uncomfortable anesthesia of the soft palate which may occur when the middle palatine nerve exists near the injection site, in addition to

hematoma which is quite rare due to the firm adherence of the palatal mucosa to the underlying bone (Malamed, 1997).

3.1.1.3 Nasopalatine Nerve Block

The nasopalatine nerve block is an invaluable technique in palatal anesthesia in which a wide area of palatal soft tissue anesthesia is achieved by a minimum volume of anesthetic solution since this technique anesthetizes both right and left nasopalatine nerves, and thus avoiding the need for multiple palatal injections and minimizing patient discomfort. However, unfortunately this technique is a highly traumatic and painful injection (Malamed, 1997).

Two approaches to this injection exist. The original technique involves one injection with a 27-gauge short needle just lateral to the incisive papilla opposite the palatal aspect of the maxillary central incisors. Insertion directly into the incisive papilla is to be avoided because the soft tissues in this area are sensitive, dense and firmly adherent to bone which increases patient discomfort. The second technique involves multiple injections and was suggested by some authors to minimize the discomfort of the original technique. The first injection is given in the labial soft tissues between the maxillary central incisors and in the second injection the needle is directed from the labial aspect through the interproximal papilla between the centrals toward the incisive papilla to anesthetize the nasopalatine nerve. Sometimes a third injection directly into the partially anesthetized palatal soft tissues overlying the nasopalatine nerve might be necessary. Although complications such as hematoma, necrosis due to high vasoconstrictor concentration, and infection after inadvertent penetration of the nasal floor might occur they are relatively rare (Malamed, 1997).

3.1.1.4 Local Infiltration of The Palate

Many patients find the palatal injections to be a very traumatic experience and many dentists as well consider the administration of palatal anesthesia to be one of the most traumatic procedures they perform in dentistry. In fact, some dentists might even warn their patients that they might feel pain or discomfort during palatal injections (Malamed, 1997).

The nerves anesthetized with this local infiltration are the terminal branches of the greater palatine and nasopalatine nerves. This injection anesthetizes the soft tissues in the immediate vicinity of the injection, and is primarily indicated for achieving hemostasis during surgical procedures, in addition to palatoginigival pain control when limited areas of anesthesia are required for operative procedures on not more than two teeth. However, it does not provide any pulpal anesthesia to the maxillary teeth. It has many advantages including acceptable hemostasis with the use of a vasoconstrictor and minimal area of numbness which reduces patient discomfort, however it is a potentially highly traumatic injection (Malamed, 1997).

A 27-gauge short needle is preferred, which is advanced to the injection site at a 45° angle and is inserted in the attached gingiva 5 to 10 mm from the free gingival margin with the bevel toward the palatal soft tissues. Anesthetic solution is deposited as the needle is advanced through the palatal mucosa until bone is gently contacted. Tissue thickness is only 2-4 mm in most areas, and consequently ischemia and blanching of the tissues will spread as the anesthetic solution is deposited. The only possible complication of this injection is necrosis of soft tissues if a highly concentrated vasoconstrictor solution is applied for hemostasis for a prolonged period of time (Malamed, 1997).

3.1.1.4.1 Justification For Palatal Injections

Extraction of any tooth requires the expansion of the alveolar bony walls to allow the tooth and root an unimpeded pathway of removal. It is also necessary to tear the periodontal ligament fibers that hold the tooth in the bony socket. To achieve this, loosening of the soft tissue attachment from the tooth, with a sharp instrument such as the elevator, is required to allow the tooth extraction forceps to be positioned more apically without impingement on the gingiva. For maxillary teeth this means the need of loosening of buccal as well as palatal soft tissues (Peterson, 2003). Therefore, for this manipulation of palatal soft tissues to be relatively pain free, anesthesia of the palatal soft tissues is required which is believed to be achieved by the palatal injection of the anesthetic solution (Malamed, 1997).

3.1.1.4.2 Techniques for Reducing The Pain of Palatal Injections

Palatal injections appear to be a painful experience for many patients (Harbert, 1989). However, it is believed that palatal injection can be administered atraumatically by many procedures such as topical anesthesia application at the injection site, pressure anesthesia at the site of injection, maintain control over the needle, slow deposition of the anesthetic solution (Malamed, 1997), application of topical ice (Harbert, 1989) and distraction techniques such as application of pressure or vibration (Budenz, 2003). Nevertheless, it is thought that the most important factor in providing an atraumatic palatal injection is the operator’s belief that it can be done painlessly (Malamed, 1997).

Furthermore, computer controlled anesthetic delivery systems may also minimize the discomfort of palatal injections, by delivering the anesthetic solution to the palatal mucosa at a rate that is potentially below the threshold of pain (Budenz, 2003).

3.1.1.4.2.1 Topical anesthesia

Surface anesthesia can be achieved by physical methods such as refrigeration anesthesia, or by pharmacological methods such as topical anesthetic agents. Properties of the ideal

topical anesthetic include that it should be non allergic, produce reliable anesthesia with sufficient duration, permit pain-free application, remain at the site of application, have an acceptable taste and do not cause any local damage or systemic toxicity (Meechan, 2000).

Topical anesthesia can be used to provide surface anesthesia of mucous membranes, the cornea or the skin. Topical anesthetics must cross tissue barriers to exert their effect, which is in contrary to injection techniques where the anesthetic solution is deposited directly in the tissues around the nerve. Therefore, topical anesthetics must either have a low pKa value to ensure high proportions of the unionized base or being used with much greater concentrations than used when injected (Lagan and McLure, 2004).

Topical anesthetics, such as 20% benzocaine gel, 5% lidocaine ointment, or 4% lidocaine topical solution, can be applied several minutes before injection to minimize the discomfort of needle insertion (Yagiela, 2000). Lidocaine is effective in concentrations 5 to 20% when used alone, the combination of 2.5% lidocaine and 2.5% prilocaine is also effective. Benzocaine is effective at concentrations of 20% when used alone and at a concentration of 15% when combined with 1.7% amethocaine (Meechan, 2002).

Topical anesthetics are used in dentistry for many purposes, such as reducing the discomfort of local anesthetic injections, decreasing the pain of operative procedures, relieving the pain of superficial mucosal lesions and anesthetizing the skin before venepuncture for sedation or general anesthesia (Meechan, 2000).

In order for topical anesthesia to be effective, tissues should be gently dried before the application of topical anesthesia (Oringer, 1949). Moreover, most topical anesthetics do not have an immediate effect. In fact, the depth of penetration of the topical anesthetic is related to the duration of application, and it seems that when applied for 5 minutes, success is guaranteed in the buccal fold of either jaw (Meechan, 2002).

The efficacy of topical anesthesia in reducing the discomfort of palatal injections has been questioned. On one hand, some authors reported that topical anesthesia reduced the discomfort of palatal injections (Svensson and Petersen, 1992, Meechan and Winter, 1996, Meechan, 2000). While on the other hand, other authors reported that it did not differ from placebo in reducing the discomfort of palatal injections (Hutchins et al, 1997, Meechan, 2000).

Some authors believe that surface preparation by topical anesthesia could reduce discomfort by two mechanisms; psychologically and pharmacologically. The psychological impact of topical anesthetics should not be disregarded because any benefit in dental practice is important (Meechan, 2002). In fact, it was found that patients who were informed that they will receive topical anesthetic for comfort anticipated less injection pain than those who were not offered such counseling, which may decrease patient apprehension (Martin et al, 1994).

On the other hand, other authors are convinced that topical anesthesia depends on the pharmacologic effect of anesthetics when applied to surface tissues. This pharmacologic effect is proved by the fact that although it is mainly used prior to injection to reduce injection discomfort, however, it can also be used alone for intraoral soft tissue surgery (Meechan, 2001) and extraction of teeth (Taware et al, 1997) or as components of preparations for symptomatic treatment of painful oral mucosal lesions such as ulcers (Meechan, 2002).

Many factors may affect the efficacy of topical anesthetics and these include the agent used, site of application and duration of application. Topical anesthetics are available in several formulations including; gels, ointments, aerosols, solutions, pastes, powders, tablets, lozenges and impregnated patches. The type of formulation can influence efficacy and therefore different formulations of the same drug require different concentrations to

achieve a similar effect (Meechan, 2002), and the concentration does affect the efficacy of topical anesthesia (Hersh et al, 1996).

The application of topical anesthetics seems to have little adverse effects on mucosa, reports of damage to mucosa are rare and any problems appear to be symptomless and reversible. In fact, concentrations of topical anesthetics up to 10 times greater than that in dental local anesthetic solutions produce plasma levels below those needed to produce toxicity. However, low doses of topical anesthetic applied to the skin of young children can produce toxicity (Meechan, 2000).

3.1.1.4.2.2 Topical Ice

Topical cooling has always been a trusted first aid remedy to reduce inflammation from trauma and to numb pain. Therefore, since cold has an overall analgesic effect, the application of topical ice on the palate before and during the injection has been suggested to relieve the discomfort of the palatal injection (Harbert, 1989).

Cooling reaches the nerves by conduction through the tissues and through the rich blood supply. The palate is supplied by three major blood arteries on each side in addition to a subepithelial network of arterioles and capillaries. However, the palate lacks the arteriovenous shunts and venae commitants which help skin resist heat loss, with the palatal vessels spreading cold more rapidly and deeply than skin (Harbert, 1989).

The effect of cold is that it slows the velocity of conduction of nerve impulses, with different types of nerve fibers experiencing a proportional decrease in velocity for each degree drop in temperature, until nerve impulses conduction stops completely in the range 0°C to 10°C. However, a waiting time of at least 5 minutes is recommended because of the thick and consequently less permeable keratinized layer of the palate. Damage to mucosa from cold was not reported and it occurs only after hours of exposure and at -2.5°C,

therefore the ice application should not be too prolonged or at a very low temperature (Harbert, 1989).

3.1.1.4.2.3 Pressure Anesthesia

Application of pressure anesthesia at the site of injection both before and during needle insertion is believed to minimize the discomfort of palatal injections. Pressure anesthesia can be produced at the site of injection by applying considerable pressure to the tissues adjacent to the injection site with a firm object such as the cotton applicator stick which is preferred or the handle of the mirror. The aim of this pressure is to produce soft tissue anesthesia through the gate control theory of pain (Malamed, 1997).

The pressure should be firm enough to produce blanching or in other words ischemia of the normally pink palatal mucosa with a feeling of intense pressure at the penetration site. Pressure anesthesia must be maintained during the needle penetration of the palatal mucoperiosteum and during the anesthetic solution deposition (Malamed, 1997).

3.1.1.4.2.4 Control Over The Needle

Control of the needle is probably of greater importance in palatal anesthesia than in any other intraoral injection. In order to achieve this control, the operator must secure a firm hand rest, so that the needle is stabilized during the palatal injection and this will reduce its discomfort (Malamed, 1997).

3.1.1.4.2.5 Slow Deposition of The Anesthetic Solution

Slow deposition of the anesthetic solution is essential in all injection techniques, not only for safety but also for providing an atraumatic injection. However, slow deposition is even of greater importance in palatal injections because of the density and firm adherence of the palatal soft tissues to the underlying bone. Rapid injection of the anesthetic solution produces high tissue pressure which tears the palatal soft tissues which causes pain on

injection and localized soreness after the anesthesia wears off, and therefore slow injection of the anesthetic solution reduces the discomfort of the injection (Malamed, 1997).

3.1.2 Extraction of Maxillary Teeth Without Palatal Injections

Anxiety is still a barrier to dental anesthesia (Nuttall et al, 2001). Fear-related behaviors are considered the most difficult aspect of patient management and can be a barrier to good care. While patients' fears may be acquired through vicarious experiences and threatening information, direct experience is the most common source of dental fear (Milgrom et al, 1997). Patients might be apprehensive about dental treatment due to fear of pain (Meechan, 2002), and ironically, local anaesthesia allows pain-free treatment and yet can elicit anxiety and is associated with many anxious thoughts and misconceptions in young patients. In fact, research has shown that 5% of the population avoids dental care because of fear of dental injections (Milgrom et al, 1997), and that dental injections are considered the most anxiety-provoking procedure for dental patients and dentists as well (Ram and Amir, 2006).

It is commonly accepted that buccal infiltration, also known as paraperiosteal field block, is usually successful when used to achieve local anesthesia for procedures on teeth or buccal soft tissue in the maxillary arch. However, palatal soft tissue anesthesia requires a separate palatal injection, a technique that is often painful for the patient (Haas et al, 1990). The pain of the palatal injection is believed to be mild to moderate and is known to be poorly tolerated by patients (Badcock et al, 2007), because the palatal mucosa is compact and tightly adherent to the underlying periosteum, in addition to its abundant nervous supply (McArdle, 1997). However, the pain of the palatal injection is believed to be caused by the displacement of the mucoperiosteum rather than the needle piercing of the mucosa (Fan et al, 2009).

In addition to the pain associated with the injection itself, palatal injections also cause numbness of the soft palate, a sensation of pressure along with unpleasant sounds. The numbness of the soft palate can be controlled to some degree by the technique of palatal anesthesia, whereas the pressure and the unpleasant sounds are largely unavoidable but of little consequence to most patients. However, it is the pain of the palatal injection that is poorly tolerated by the patient (Badcock et al, 2007). In fact, palatal injections are rated as one of the most painful procedures in dentistry and its direct experience was found to be the most common source of fear of dental surgery (Fan et al, 2009, Milgrom et al, 1997).

The discomfort associated with palatal injections is a concern to most dentists, many of whom avoid using them unless they are absolutely necessary (Fan et al, 2009). It has been shown that for the dentist the administration of the palatal injection is rated as one of the most traumatic procedures in dentistry. In fact, many dental professionals advice their patients that they will experience pain rather than discomfort before the administration of palatal injections and this "forewarning" allows the patient to become psychologically prepared and relieves the operator of responsibility when the pain occurs (Badcock et al, 2007).

Most dentists try to reduce the pain of palatal injections, however the most commonly used techniques offer little if any proven benefit, and even though a number of modern injection techniques may be used to reduce the discomfort of palatal injections, however, there is little evidence that these methods are universally effective, and palatal injection remains a painful experience for most patients (Fan et al, 2008).

Application of topical anesthesia has been a frequently used option to reduce the discomfort of palatal injections despite its effectiveness in the palate being questioned, mainly because it is effective only on surface tissues whereas tissues deep to the area of application are poorly anesthetized, and although this surface anesthesia allows an

atraumatic needle penetration, the density of the palatal mucosa and its firm adherence to the underlying bone still causes pain during palatal injections. EMLA has been found to be more effective than conventional topical anesthetics and has been used effectively intraorally, however, it is not made for intraoral administration and does not contain a flavoring agents which makes it bitter tasting. Transcutaneous electronic nerve stimulation has also been suggested for alleviating the pain of palatal injections, however, its use is not simple with probes that need to be held in position manually for 2 minutes. Despite the usefulness of these techniques, they are expensive, complex and time-consuming procedures and patients still experience discomfort during palatal injections (Uckan et al, 2006).

Furthermore, the "chasing technique" has also been suggested to avoid the discomfort of palatal injections where the palatal tissues are approached through the interdental papilla after the initial buccal injection. In addition, some dentists believe that the use of a 30-gauge needle for administration of the palatal injection diminishes its pain, however it has been reported that this technique does not offer any advantage over 27- or even 25-gauge needles. In fact, Malamed stated that there is no specific recommendation for the use of a 30-gauge needle for palatal injections (Badcock and McCullogh, 2007).

The routine use of palatal injections is based on the anatomical description of the sensory innervations of the palate which dictates, as conventionally taught to dental practitioners, anesthetizing these innervations by a separate palatal injection for procedures involving manipulation of the palatal soft or hard tissues, including extraction of maxillary teeth (Badcock et al, 2007). The administration of a separate palatal injection may be avoided by blocking the maxillary nerve or the greater palatine nerve in the pterygopalatine fossa. However, the technical difficulty and the potential morbidity of such techniques have prevented their routine acceptance (Blanton and Jeske, 2003, Badcock et al, 2007).

A survey in Australia and New Zealand have shown that 8.3% of the oral and maxillofacial surgeons over there occasionally removed maxillary third molars without palatal injections, with one surgeon reported the extraction of maxillary third molars without ever administering a palatal injection, and this fact does suggest that palatal injection might not ever be required for extraction of maxillary third molars. Therefore, Badcock and McCullough suggested that the requirement of the palatal injection for the removal of the maxillary third molars may not be absolute as conventionally taught, which demonstrates the need for further investigation of this possibility (Badcock and McCullough, 2007).

The avoidance of palatal injections completely when they are not necessary is desirable, (Badcock et al, 2007), since offering comfortable palatal anesthesia to the patient represents a practice building strategy that decreases the patient's anxiety and increases treatment acceptance (McArdle, 1997).

Any local anesthetic that would permit use of buccal infiltration to gain palatal anesthesia would be of great advantage in dentistry (Haas et al, 1990), since the reputation of palatal injections seems to persist even though guidelines for gentler procedures have been suggested (Harbert, 1989). The relatively new local anesthetic articaine has been claimed to achieve anesthesia of mandibular pulpal and lingual soft tissue by labial or buccal infiltration, as well as palatal soft tissue anesthesia by means of maxillary labial or buccal infiltration. This would be of important clinical benefit as it is in contrast to commonly used local anesthetics which are efficacious by infiltration for labial soft tissue and maxillary pulpal anesthesia only (Haas et al, 1990).

Many practitioners have found that palatal injections might not be necessary after maxillary buccal infiltration of 4% articaine (Budenz, 2003). Decades ago, Frenkel commented on the "extraordinary depth" of anesthesia achieved with articaine by stating that even extractions of mandibular premolars could be done with buccal infiltration only

(winther and patirupanusara, 1974). In addition, Corbett et al (2008) have found that some patients reported lingual mucosa numbness after only buccal infiltration with articaine in the mandibular sulcus.

3.1.2.1 Basic Concept

Many studies showed that extraction of maxillary teeth was possible with articaine buccal infiltration only (Uckan et al, 2006, Fan et al, 2009, Badcock et al, 2007), which was believed to be achieved by transalveolar diffusion of the buccally administered local anesthetic agent (Corbett et al, 2008). It is believed that the relatively porous thin bone of the buccal maxilla facilitates the diffusion of any local anesthetic (Fan et al, 2009). It has been claimed that the diffusion of articaine through soft and hard tissues is better than other local anesthetics and that maxillary buccal infiltration of articaine provides palatal soft tissue anesthesia, obviating the need for a palatal injection (Uckan et al, 2006).

In fact, some dentists reported that for extraction of maxillary third molars, a waiting of at least one minute after administering buccal infiltration and before the palatal injection will allow the diffusion of the local anesthetic solution to anesthetize the palatal tissues before the injection. With this concept in mind, omitting the palatal injection in favor of this palatal anesthesia might be possible (Badcock and McCullough, 2007).

In addition, other investigators believed that the anesthetic requirement for tooth extraction is not as high as that required for routine conservative dental treatment and this fact facilitates extraction of maxillary teeth without the painful palatal injection (Fan et al, 2009).

3.1.2.2 Previous Trials

Li has described the extraction of maxillary teeth without a separate palatal injection by blocking the palatal nerves in the pterygopalatine fossa (Badcock et al, 2007). Even though this technique avoids the painful palatal injection, blocking palatal nerves in the

pterygopalatine fossa is technically difficult and associated with high morbidity (Blanton and Jeske, 2003, Badcock et al, 2007).

Uckan et al (2006) found that buccal infiltration with a protracted latency period was superior to all the aforementioned techniques of reduction of the pain of palatal injections in permanent maxillary tooth extraction. They found that the pain of maxillary teeth removal without palatal injection was similar to their removal with palatal injection, and concluded that the removal of maxillary teeth without palatal injection is possible by articaine buccal infiltration (Uckan et al, 2006).

Badcock et al (2007) suggested that the routine use of palatal injections for the extraction of the maxillary third molars has never been validated, and reported the clinical equivalence of lidocaine and normal saline when injected in the palate for the extraction of maxillary third molars. They concluded that the routine use of palatal injection of local anesthesia for extraction of maxillary third molars is difficult to justify and may not be required (Badcock et al, 2007). Lima-Júnior et al (2009) also reported that impacted maxillary third molars extraction can be performed with only buccal infiltration of articaine without the need of a palatal injection.

Lassemi et al (2008) found that labial infiltration can be an effective alternative to the conventional palatal infiltration for effective anesthesia of the anterior palate by inserting the needle superior to the apices of central incisors in the area of the superior border of the base of the anterior nasal spine near the nasal floor. They have found that the use of this technique without palatal injection provides sufficient anesthesia for the extraction of maxillary central incisors (Lassemi et al, 2008).

Fan et al (2009) compared extraction of maxillary teeth after only buccal infiltration and their extraction with buccal and palatal injections. They found that the routine use of buccal and palatal injections causes more discomfort to the patient than the use of buccal

infiltration alone. Whereas there was no significant difference in extraction pain between the two injection techniques, and thus they reported that the buccal infiltration with articaine provided similar clinical efficacy to the routine anesthesia with palatal injection, and thus the use of palatal injection might not be required for the extraction of maxillary teeth (Fan et al, 2009).

Peng et al (2008) also compared the extraction of maxillary teeth with palatal injection and their extraction without palatal injection and found that there was no significant difference between the two groups. They concluded that extraction of maxillary teeth without the administration of palatal injection can be achieved by articaine buccal infiltration (Peng et al, 2008).

All the aforementioned studies showed that the extraction of maxillary teeth without palatal injection is possible by the buccal administration of articaine, although further investigation is needed to demonstrate this possibility for lidocaine as well, in addition, to comparing the anesthetic efficacy of articaine with lidocaine when they are administered as buccal infiltration for the extraction of permanent maxillary teeth.

Chapter four: Materials and Methods

4.1 Study Design

The study was a single blinded clinical trial with randomization of matched pairs design, which included two control groups and one experimental group and where the effect of using different local anesthetic agents and different anesthetic techniques on the patient's perception of pain during local anesthetic injections and maxillary teeth extraction were compared between the three groups.

4.2 Ethical Considerations

The protocol of the study was approved by the Institutional Review Board (IRB) of Jordan University of Science and Technology.

4.3 Study Sample and Settings

The study population consisted of 131 patients, 47 males and 84 females with a mean age of 30.6 years that ranged from 13 to 62 years, 23 patients had more than one tooth to be extracted with 16 patients acting as their own control. Patients were recruited from the oral surgery department in the dental teaching center of the Jordan University of Science and Technology. The patients were referred for extraction of their permanent maxillary teeth from different departments of the center after it was decided that extraction was the suitable treatment for these patients. The patients who approved to be included in the study were allocated into three groups which differed in the administered local anesthetic agent and the area of administration, and were matched according to gender, age, smoking status, tooth to be extracted and the reason for extraction.

The first group was the positive control group which received buccal and palatal local anesthetic injections of 2% lidocaine with 0.015mg/ml epinephrine (Xylestesin-A®, 3M ESPE, Germany). The second group was the negative control group which received only buccal local anesthetic injection of 2% lidocaine with 0.015mg/ml epinephrine (Xylestesin-A®, 3M ESPE, Germany). The third group was the experimental group which received only buccal injection of 4% articaine with 0.012mg/ml epinephrine (Ubistesin Forte®, 3M ESPE, Germany).

The dental teaching center of faculty of dentistry at the Jordan University of Science and Technology was the setting of the study. This center is located in Irbid in North Jordan and is a place for the training of undergraduate dental students and internship trainees. It provides free dental services for northern Jordan community.

4.3.1 Inclusion Criteria

Only individuals meeting all the following criteria were included in the study:

1. Age equal or above 13 years.
2. Maxillary tooth that is indicated for simple extraction under local anesthesia.
3. Nonsurgical extraction of the tooth.
4. Tooth is not periodontally involved.
5. No history of any medical condition that may affect the patient’s perception of pain.

4.3.2 Exclusion Criteria

Any patient with any of the followings was excluded from the study:

1. Children under the age of 13 years.
2. Surgical extraction
3. Mobility or periodontal involvement of the tooth to be included in the study.
4. Emergency extraction due to severe pain.
5. The presence of symptomatic infection such as periapical or vestibular abscess.

6. The presence of a history of any medical condition that may affect the patient's perception of pain.

7. Inability to understand the visual analogue scale or the verbal response scales.

8. Presence of suspected allergy to either lidocaine or articaine.

4.4 Procedures

4.4.1 Patient History

Following recruitment and after checking the patient's referral papers to the oral surgery department and obtaining the informed consent to participate in the study (Appendix A), each patient was interviewed by the researcher using especially designed forms (Appendix B) for collecting the following data; demographic data (name, gender, age, occupation, marital status, address and phone number), medical history, medications, oral hygiene practices, dental history with specific questioning about previous extractions in the maxilla and social history which included cigarette smoking frequency and amount of cigarettes smoked per day. The classification of patients into smokers or nonsmokers was based on the amount of their smoking as number of cigarettes per day, with patients smoking 5 or more cigarettes per day considered as smokers.

4.4.2 Extraoral and Intraoral Examination

Patients were examined intraorally and extraorally. Tooth in question was examined clinically and radiographically for its general condition and periodontal condition including the presence of any mobility, bone resorption or infection.

4.4.3 Administration of Local Anesthesia

Each patient received local anesthesia according to his/her group. However, prior to the administration of the local anesthetic each patient had an explanation of the study aims and

procedures along with the visual analogue and verbal response scales by the researcher and whenever possible, patients were blinded to the anesthetic agent and technique used.

Local anesthesia was administered with a 30-gauge short needle. For buccal supraperiosteal injection, after retracting the lip or the cheek and pulling the tissues tight and with the syringe parallel to the long axis of the tooth, the needle was inserted in the height of the mucobuccal fold above the apex of the tooth to be extracted and advanced 2 to 3 mm with the bevel facing the bone. The anesthetic solution was then injected with the needle not touching the bone to minimize pain to the patient. For the local infiltration of the palate, needle was advanced to the site at a 90° angle to mucosal surface and was inserted in the palatal mucosa midway between the palatal gingival margin of the tooth to be extracted and the midline of the palate with the bevel toward the palatal soft tissues. Anesthetic solution was deposited as the needle was advanced through the palatal mucosa until bone was gently contacted. The rate of injection for all anesthetic techniques was about 1ml/minute. All local anesthetic injections were administered by the same operator who was not blinded to the administered local anesthetic agent.

The number of local anesthetic injections and the amount of local anesthetic solution needed to complete the extraction were recorded in terms of dental cartridges. The protocol of the study included the administration of three-quarters a cartridge buccally and one-quarter a cartridge palatally for group A, 1 cartridge as a second buccal injection for group A when needed, 1 cartridge as first buccal injection for group B and C, three-quarters a cartridge as second buccal injection when needed for group B and C, one-quarter a cartridge as a palatal injection when needed for group B and C.

4.4.4 Assessment of Injection Pain

Three scales were used for the measurement of injection pain; one visual analogue scale and two verbal response scales. The visual analogue scale consisted of a 100 mm

ungraduated continuous horizontal line anchored with two end points labeled “no pain at all” on the left and “unbearable pain” on the right.

After local anesthetic injection, each patient was asked to mark the line of the visual analogue scale according to the intensity of pain that was experienced during the injection, and the distance from the left end of the scale to the patient’s mark was used as a numerical index of the severity of pain experienced.

The first verbal response scale was named VRS1 and included three categories; mild pain, moderate pain and severe pain from which the patient chose the category that was most representative of his/her pain. The second verbal response scale was named VRS2 and also was composed of three categories in which each patient was asked to describe the level of pain as less than expected, as expected or greater than expected. In all cases the visual analogue scale was completed first.

4.4.5 Extraction Procedure

A standard procedure was followed in this study for calculating and recording the amount of administered local anesthesia and waiting time before beginning the extraction. For group A, the standard protocol included the administration of three-quarter the cartridge buccally and one-quarter palatally. The extraction was started immediately after the local anesthetic administration. For group B and C, the first buccal anesthetic injection was administered as one cartridge, and then a waiting period of 5 minutes was allowed for the anesthetic effect. The extraction was then attempted with either forceps or an elevator with the patients being periodically questioned if they felt pain. If an unacceptable level of pain or discomfort was reported by the patient, the extraction was aborted and another 5 minutes of waiting were allowed. After that another attempt to extract the tooth was done and if the patient reported pain a third waiting period of 5 minutes was allowed. An extraction attempt followed and if the patient reported pain then a second buccal injection of three-

quarter cartridge was administered after which a 5 minute waiting period was allowed and the extraction was then attempted. Again if the patient reported pain, another 5 minutes waiting period was allowed and then extraction was attempted, and if pain was reported another 5 minutes waiting period was allowed after which extraction was again attempted and this time if pain was reported a palatal injection of one-quarter cartridge was administered and recorded and the patient was asked to assess the pain of this palatal injection by the aforementioned scales.

All extractions were performed by the researcher. After the extraction, patients were asked to rate the severity of pain that was experienced during the extraction using the visual analogue scale and the verbal response scales. In addition, the following data were recorded for every patient: the time of extraction which was calculated in minutes from the first relatively painless application of the forceps or the elevator until the complete removal of the tooth, waiting time which was also calculated in minutes from the administration of the first cartridge until the ability of the operator to start the extraction with minimal or no discomfort to the patient, tooth that was extracted and reason of extraction as judged from the referral papers and by the researcher. In addition any intraoperative complication that was encountered during the extraction procedure was recorded.

4.5 Statistical Analysis

Data were numerically coded, grouped and analyzed using Statistical Package for Social Sciences (SPSS) software (version 15.0. SPSS®: Inc., Chicago, IL, USA). Descriptive statistics, one way ANOVA, t-test, independent t-test, chi-square test and pearson test were used as appropriate. Significance level was set at $P < 0.05$.

Chapter five: Results

The overall study sample consisted of 131 patients; 47 males and 84 females and their age ranged from 13 to 62 years. Twenty-three patients had more than one tooth to be extracted with 16 patients acting as their own control. Patients were allocated into 3 different groups and matched according to their age, gender, smoking status, tooth to be extracted and reason for the extraction. The first group was the positive control group which was named "Group A" and consisted of 55 patients who received buccal and palatal local anesthetic injections of 2% lidocaine with 0.015mg/ml epinephrine (Xylestesin-A®, 3M ESPE, Germany). The second group was the negative control group which was named "Group B" and consisted of 50 patients who received only buccal local anesthetic injections of 2% lidocaine with 0.015mg/ml epinephrine (Xylestesin-A®, 3M ESPE, Germany). The third group was the experimental group which was named "Group C" and consisted of 50 patients who received only buccal injection of 4% articaine with 0.012mg/ml epinephrine (Ubistesin Forte ®, 3M ESPE, Germany).

5.1 Description of Recruitment Variables

5.1.1 Gender

A total of 47 males participated in the study who were distributed as follows; 20 males were included in group A representing 36.40% of the group, 15 males in group B representing 30.00% of the group and 16 males in group C representing 32.00% of the group. On the other hand, a total of 84 females participated in the study who were distributed as 35 females in group A representing 63.60% of the group, 35 females in group B representing 70.00% of the group and 34 females in group C representing 68.00%

of the group (Figure 5.1). The differences in gender distribution between the three groups were statistically insignificant (P = 0.78).

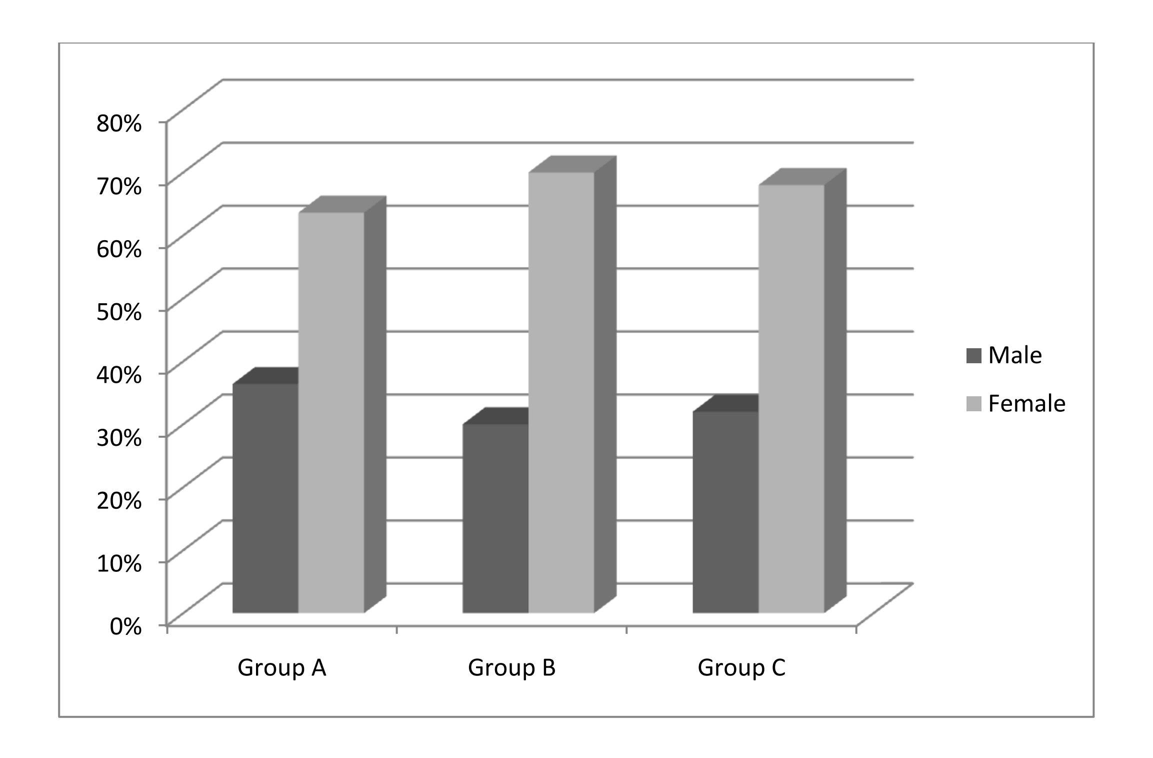


Figure 5.1: Distribution of the study groups according to gender

5.1.2 Age

The age of patients in group A ranged from 13 to 62 years with a mean of 30.35 (± 9.52) years, whereas in group B it ranged from 13 to 57 years with a mean of 30.18 (± 10.58) years and in group C it ranged from 13 to 62 years with a mean of 31.36 (±10.21) years (Figure 5.2). The differences in the mean age between the three groups were statistically insignificant (P = 0.82).

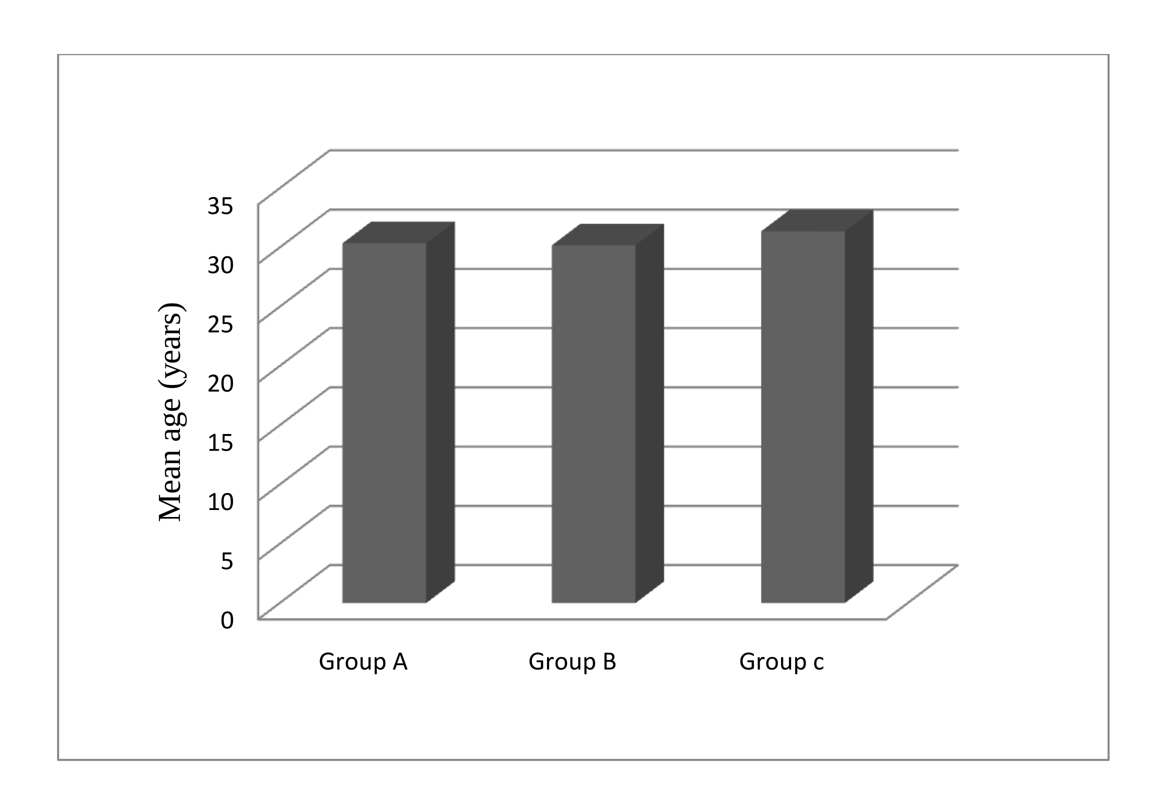


Figure 5.2: Mean age of the three study groups

5.1.3 Previous extraction in the maxilla

Of the whole study population 67 patients experienced extraction in the maxilla for the first time who were distributed as 24 patients in Group A representing 43.60% of the group, 22 patients in group B representing 44.00% of the group and 21 patients in group C representing 42.00% of the group. On the other hand, 88 patients had previous extractions in the maxilla who also included patients having second time extraction as their own control or as having more than one tooth to be extracted. These patients were distributed as 31 patients in group A representing 56.40% of the group, 28 patients in group B representing 56.00% of the group and 29 patients in group C representing 58.00% of the group (Figure 5.3). The differences in the previous extraction experience between the three groups were statistically insignificant (P = 0.98).

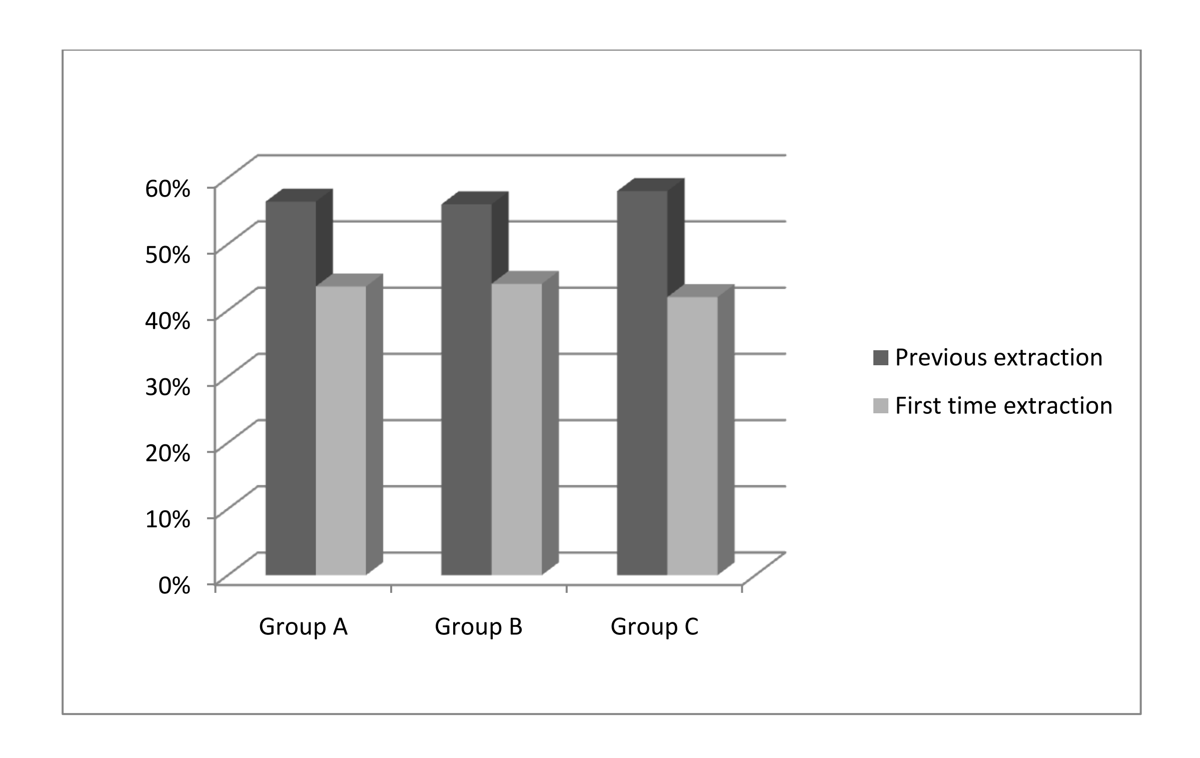


Figure 5.3: Comparison of previous extraction experience in the maxilla between the three groups.

5.1.4 Smoking Status

In group A 16 patients were smokers representing 29.10% of the group. In group B 12 patients were smokers who represented 24.00% of the group and in group C 12 patients were smokers who represented 24.00% of the group. Non smokers were distributed as 39 patients in group A representing 70.90% of the group, 38 patients in group B representing 76.00% of the group and 38 patients in group C representing 76.00% of the group (Figure 5.4). The differences between the three groups according to smoking status were statistically insignificant (P = 0.79).

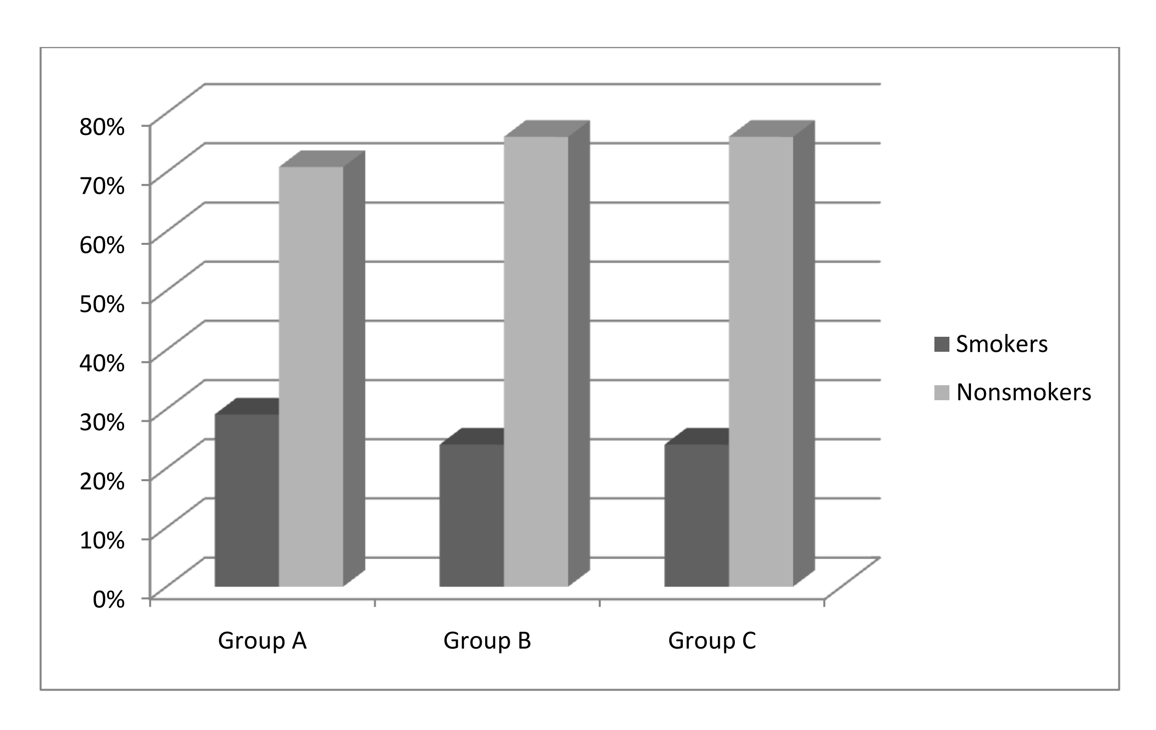


Figure 5.4: Distribution of the study groups according to smoking status

The amount of smoking was recorded for each patient as the number of cigarettes smoked per day. For group A the amount of smoking ranged from 0 to 60 cigarettes/day with a mean amount of 7.62 (± 13.85) cigarettes/day. For group B the amount of smoking ranged from 0 to 60 cigarettes/day with a mean amount of 5.78 (±12.87) cigarettes/day and for group C the amount of smoking ranged from 0 to 40 cigarettes/day with a mean amount of 4.80 (± 9.47) cigarettes/day (Figure 5.5). The differences in the mean amount of smoking between the three groups were statistically insignificant. (P = 0.49)

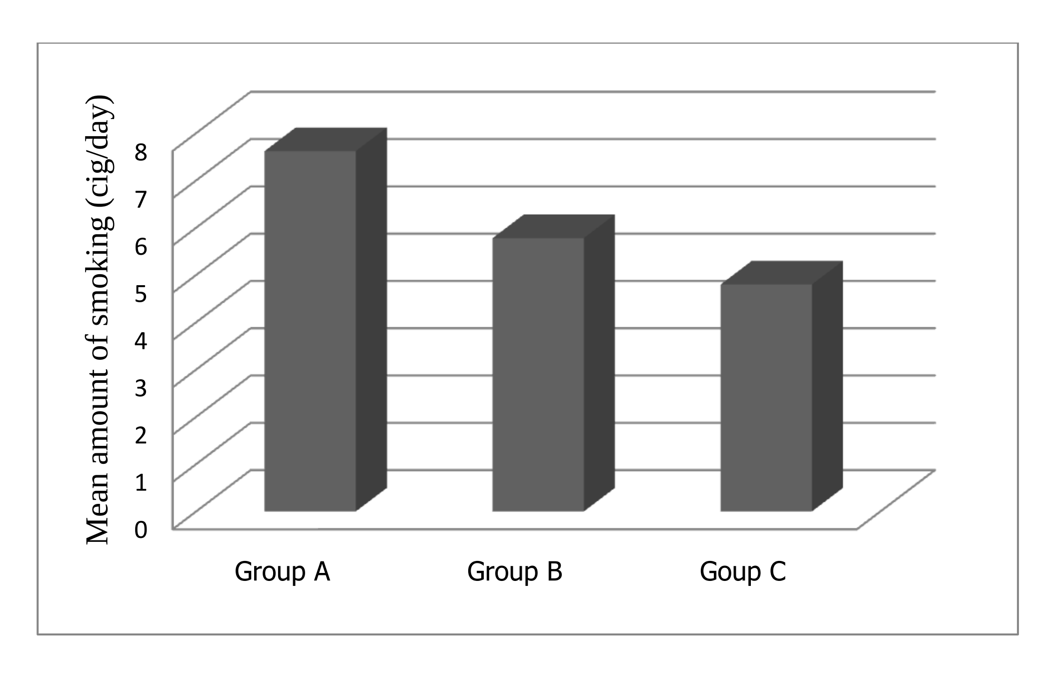


Figure 5.5: Mean amount of smoking in the study groups

5.1.5 Extracted Teeth

A total of 155 permanent maxillary teeth were extracted in the study which included 1 central incisor, 3 lateral incisors, 21 first premolars, 28 second premolars, 15 first molars, 17 second molars and 70 third molars (Figure 5.6). The central incisor was extracted in group A and represented 1.80% of extracted teeth in the group. For lateral incisors, 2 were extracted in group A and represented 3.60% of extracted teeth in the group and 1 in Group B which represented 2.00% of extracted teeth in the group. For first premolars, 8 were extracted in group A representing 14.50% of extracted teeth, 5 were extracted in group B representing 10.00% of extracted teeth and 8 were extracted in Group C representing 16.00% of extracted teeth. For second premolars, 7 were extracted in group A representing 12.70% of extracted teeth, 10 were extracted in group B representing 20.00% of extracted teeth and 11 were extracted in group C representing 22.00% of extracted teeth. For first molars, 6 were extracted in group A representing 10.90% of extracted teeth, 5 were extracted in group B representing 10.00% of extracted teeth and 4 were extracted in group C representing 8.00% of extracted teeth. For second molars, 8 were extracted in group A

which represented 14.50% of extracted teeth, 7 were extracted in group B representing 14.00% of extracted teeth and 2 in group C representing 4.00% of extracted teeth. For third molars, 23 were extracted in group A representing 41.80% of extracted teeth, 22 were extracted in group B representing 44.00% of extracted teeth and 25 were extracted in group C which represented 50.00% of extracted teeth (Figure 5.7). The difference in the distribution of different teeth between the three groups was statistically insignificant (P = 0.65).

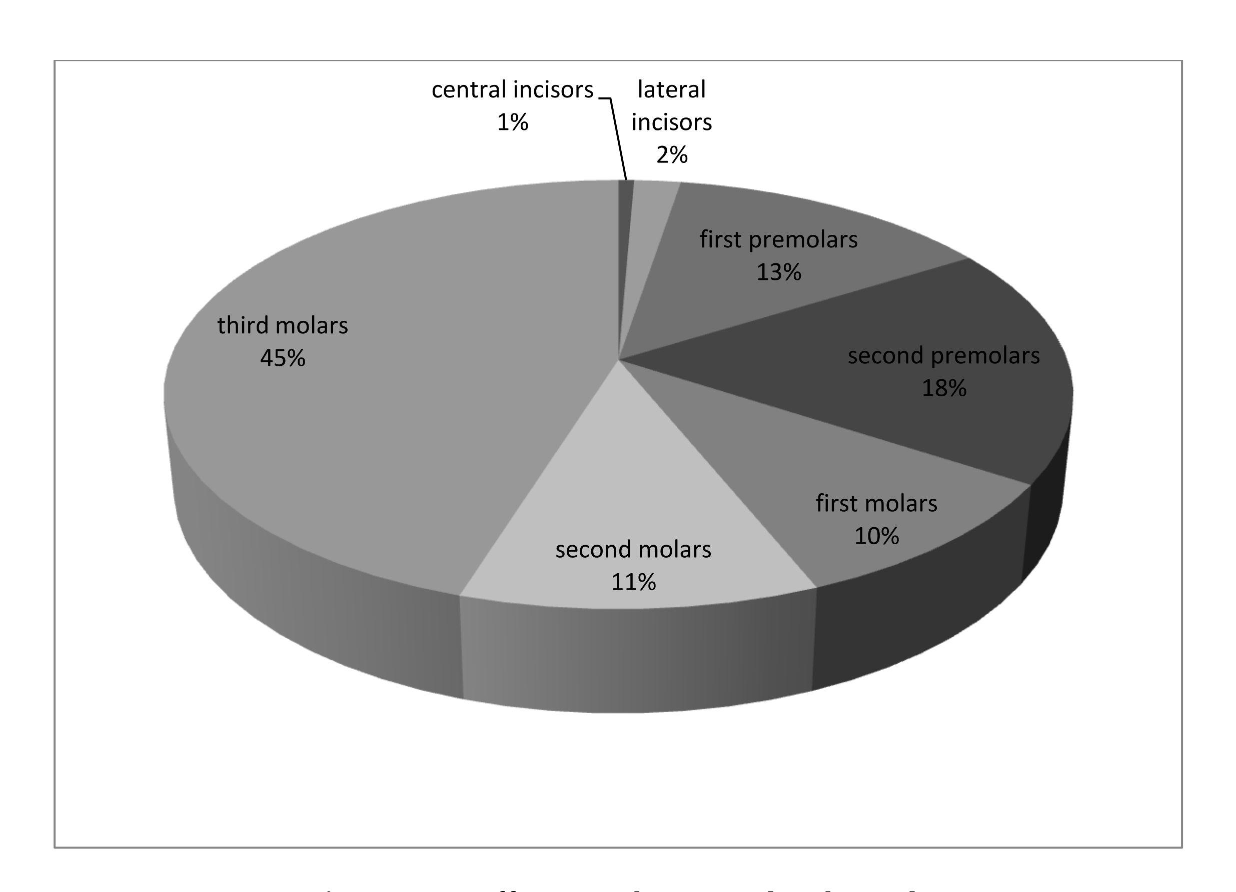


Figure 5.6: Different teeth extracted in the study

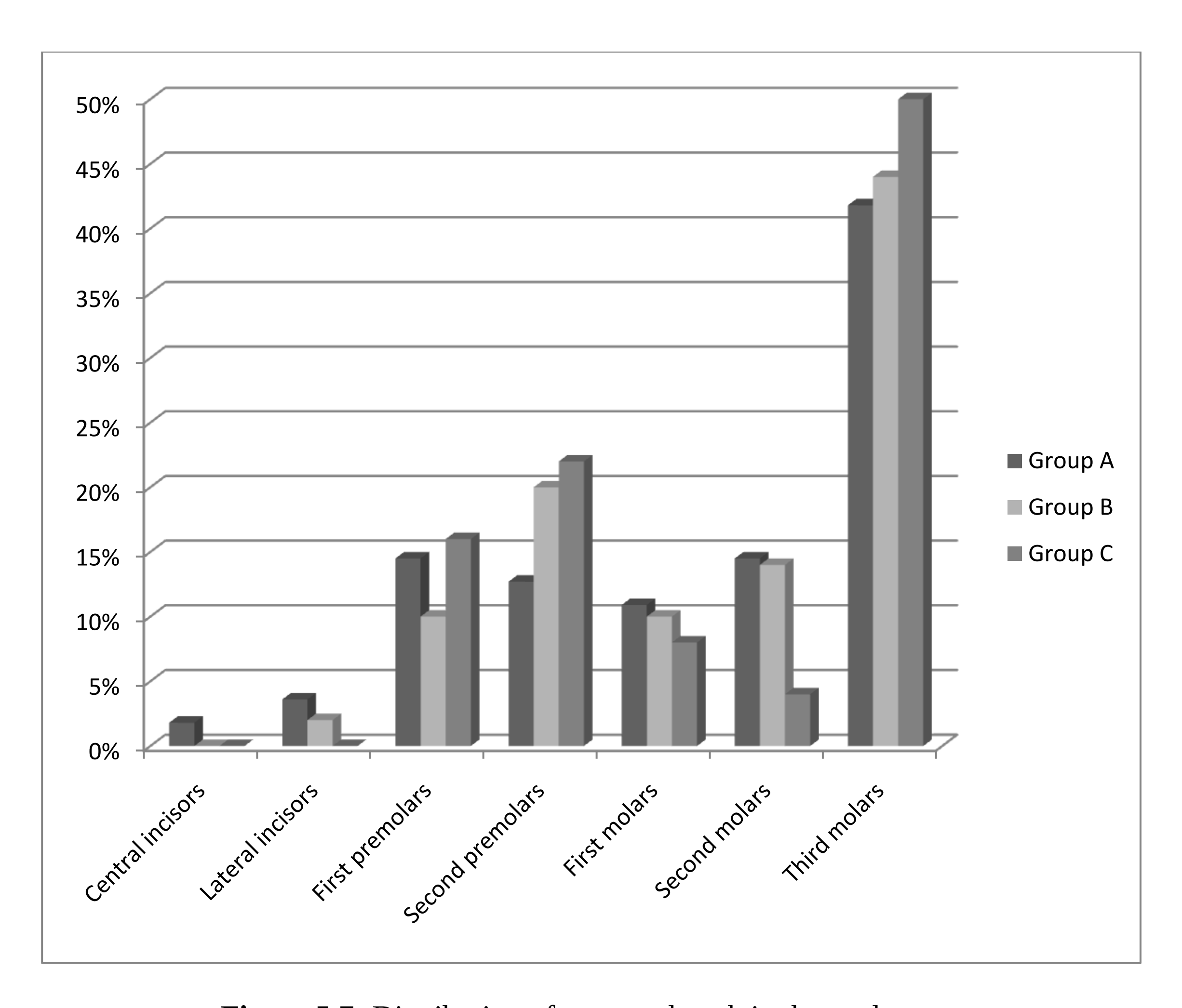


Figure 5.7: Distribution of extracted teeth in the study groups

5.1.6 Reason of Extraction

Reasons for extraction of teeth included caries, orthodontic reasons, cheek biting, pericoronitis, prosthodontics reasons, endodontic failure, nonfunctional third molars and remaining roots. Due to caries, 27 teeth were extracted in group A representing 49.09% of extracted teeth, 28 teeth were extracted in group B representing 56.00% of extracted teeth and 23 teeth were extracted in group C representing 46.00% of extracted teeth. For orthodontic reasons, 6 teeth were extracted in group A representing 10.90% of extracted teeth, 5 teeth were extracted in group B representing 10.00% of extracted teeth and 6 teeth were extracted in group C representing 12.00% of extracted teeth. Due to cheek biting, 6 teeth were extracted in group A representing 10.90% of extracted teeth, 4 teeth were

extracted in group B representing 8.00% of extracted teeth and 7 teeth were extracted in group C representing 14.00% of extracted teeth. For being remaining roots, 15 teeth were extracted in group A representing 27.30% of extracted teeth, 11 teeth were extracted in group B representing 22.00% of extracted teeth and 12 teeth were extracted in group C representing 24.00% of extracted teeth. Due to pericoronitis, 1 tooth was extracted in group A representing 1.80% of extracted teeth. Due to endodontic failure 1 tooth was extracted in group B representing 2.00% of extracted teeth and due to prosthodontics reasons 1 tooth was extracted in group B representing 2.00% of extracted teeth. For being nonfunctional third molars 2 teeth were extracted in group C representing 4.00% of extracted teeth (Figure 5.8). The differences in the distribution of the reasons of extraction among the three groups were statistically insignificant ($P = 0.61$).

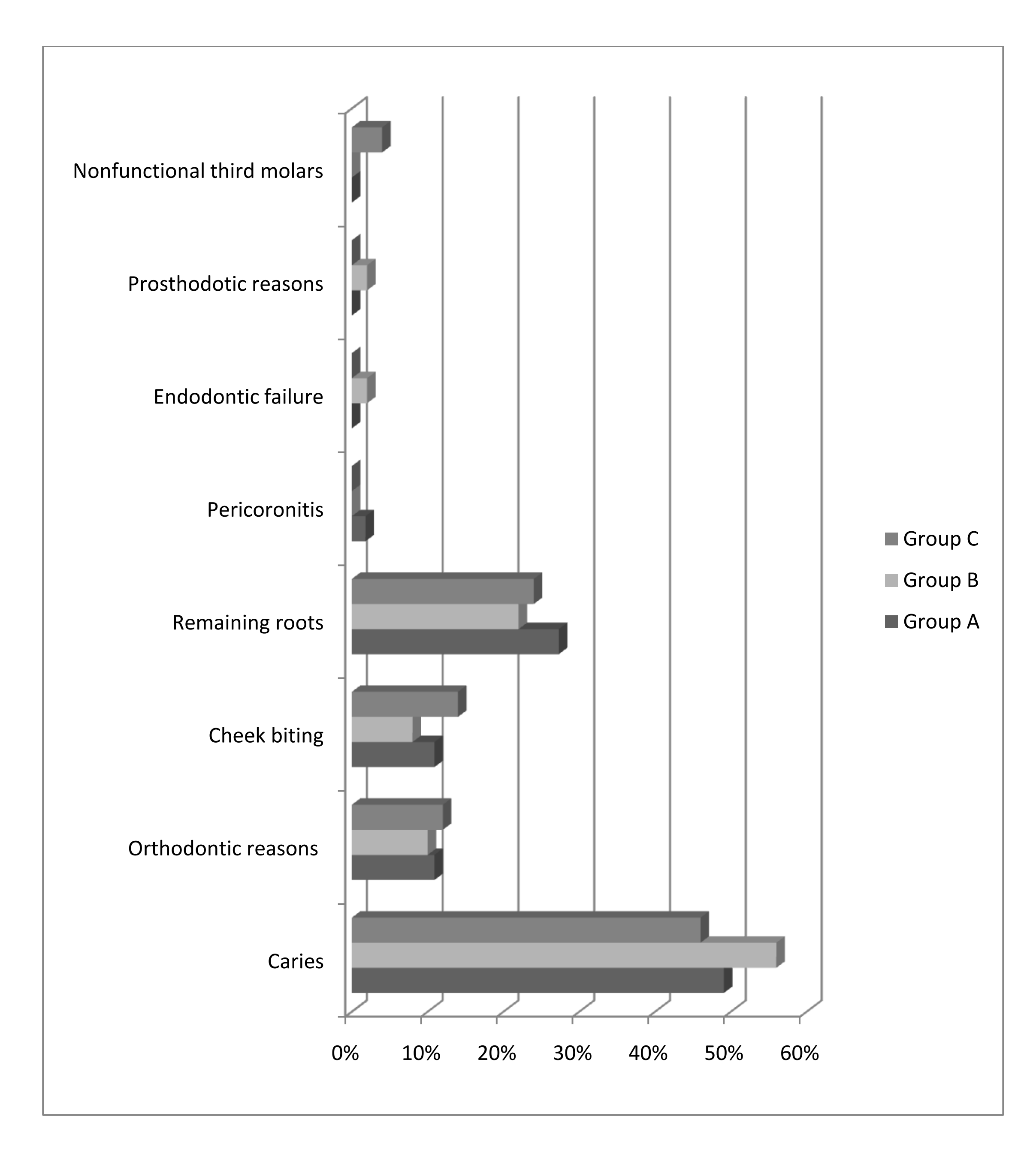


Figure 5.8: Distribution of reasons for extraction in the study groups.

5.2 Description of Procedure Variables

5.2.1 Intraoperative Complications

Intraoperative complications that were encountered during the extraction procedure included fracture of the tooth whether fracture of the crown or of the root, fracture of maxillary tuberosity, trauma to soft tissues, luxation of an adjacent tooth, pain and

oroantral communication. Fracture of the tooth was encountered in 14 extractions in group A representing 25.50% of the group, in 16 extractions in group B representing 32.00% of the group and in 10 extractions in group C representing 20.00% of the group. Fracture of maxillary tuberosity was encountered in 1 extraction in group A representing 1.80% of the group and in 2 extractions in group B representing 4.00% of extracted teeth. Fracture of the tooth and trauma to soft tissues were encountered in 1 extraction in group A representing 1.80% of extractions in the group and in 1 extraction in group C representing 2.00% of extractions in the group. Fracture of the tooth and the maxillary tuberosity were encountered in 1 extraction in group C representing 2.00% of extractions in the group.

Pain during the extraction was encountered in 2 extractions in group A representing 3.60% of extractions in the group, in 1 extraction in group B representing 2.00% of extractions in the group and in 2 extractions in group C representing 4.00% of extractions in the group. Fracture of the tooth along with pain was encountered in 1 extraction in group B representing 2.00% of extractions in the group and in 1 extraction in group C representing 2.00% of extractions in the group. Luxation of an adjacent tooth was encountered in 1 extraction in group C representing 2.00% of extractions in the group and oroantral communication was also encountered in 1 extraction in group C representing 2.00% of extractions in the group. No complications were encountered in 37 extractions in group A representing 67.30% of extractions in the group, 30 extractions in group B representing 60.00% of extractions in the group and 33 extractions in group C representing 66.00% of extractions in the group (Figure 5.9). The differences in the occurrence of intraoperative complications between the three groups were statistically insignificant (P = 0.72).

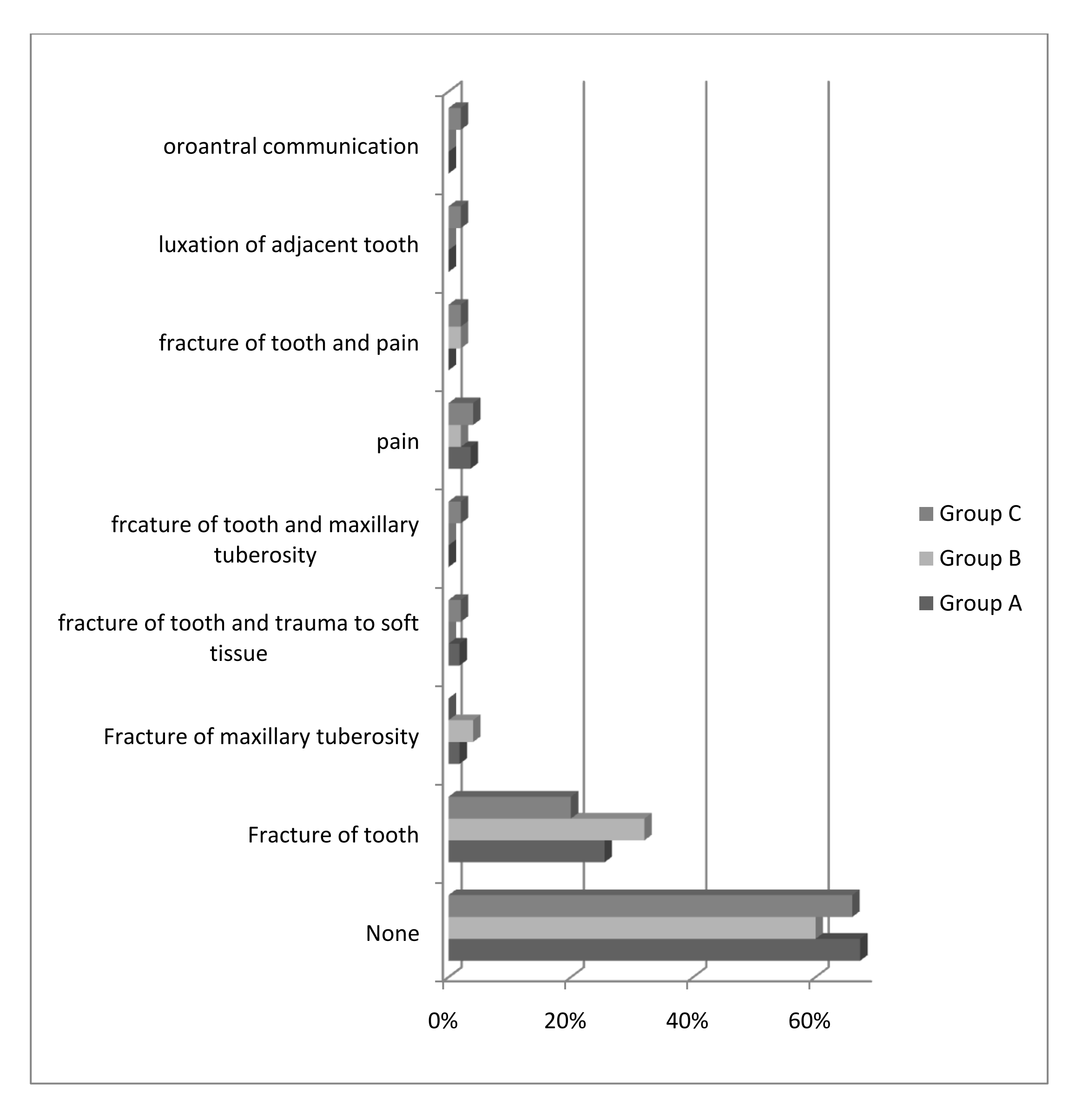


Figure 5.9: Intraoperative complications in the three study groups

5.2.2 Amount of Buccally Administered Local Anesthesia

The amount of buccally administered local anesthesia was recorded for each patient in terms of the number of cartridges or fractions of the cartridge as approximated to the nearest ¼ of that was administered until complete removal of the tooth. The amount of buccally administered local anesthesia for group A ranged from 0.75 to 1.75 cartridges with a mean amount of 0.86 (± 0.26) cartridges. The amount for group B ranged from 1.00 to 2.00 cartridges with a mean amount of 1.34 (± 0.44) cartridges and for group C the amount also ranged from 1.00 to 2.00 cartridges with a mean amount of 1.32 (± 0.44)

cartridges (Figure 5.10). The differences in the amount of local anesthesia administered buccally between the three groups were statistically significant (P < 0.01). On comparing each two groups separately it was found that the difference between group A and group B was statistically significant ($P < 0.01$) and the difference between group A and group C was also statistically significant ($P < 0.01$). However, the difference between group B and Group C was statistically insignificant. ($P = 0.82$)

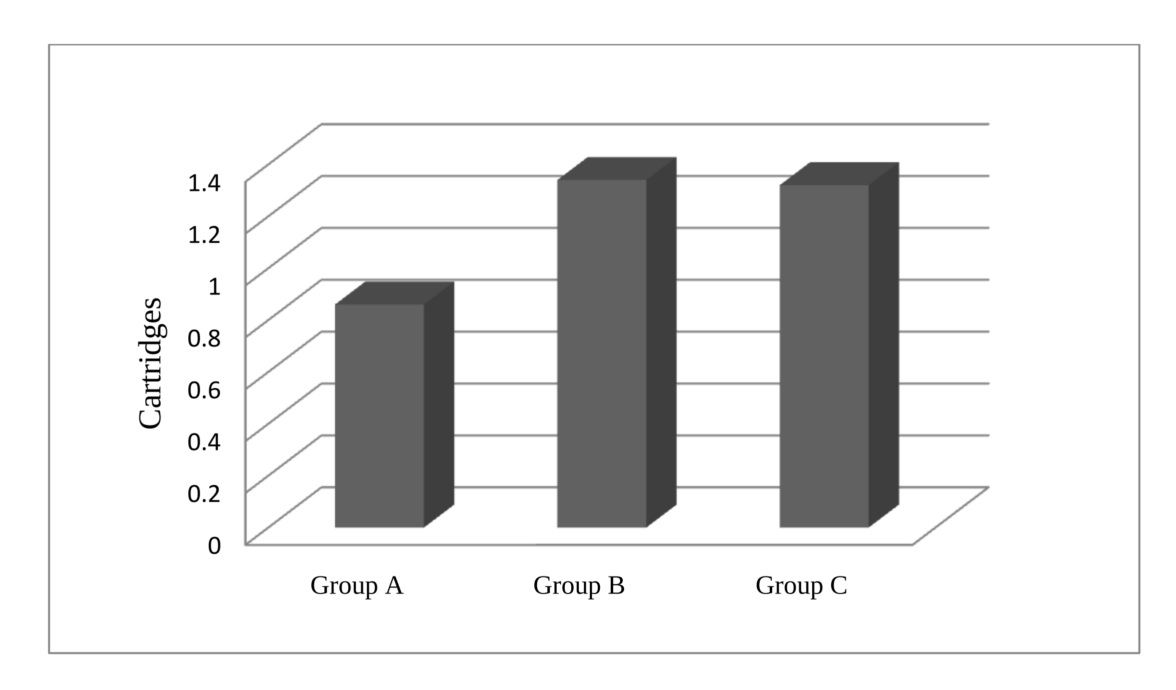


Figure 5.10: Mean amount of buccally administered local anesthesia in the three study groups

5.2.3 Waiting Time

Waiting time was recorded for patients in group B and group C. It was calculated in minutes from immediately after the administration of the first cartridge until the ability of the operator to start the extraction with minimal or no pain to the patient and was only calculated for groups B & C. For group B, the waiting time ranged from 5.00 to 15.00 minutes with a mean of 9.60 (± 3.76) minutes and for group C, it ranged from 5.00 to 30.00

minutes with a mean of 11.50 (± 5.91) minutes (Figure 5.11). The difference in the mean waiting time between the two groups was statistically insignificant (P = 0.06).

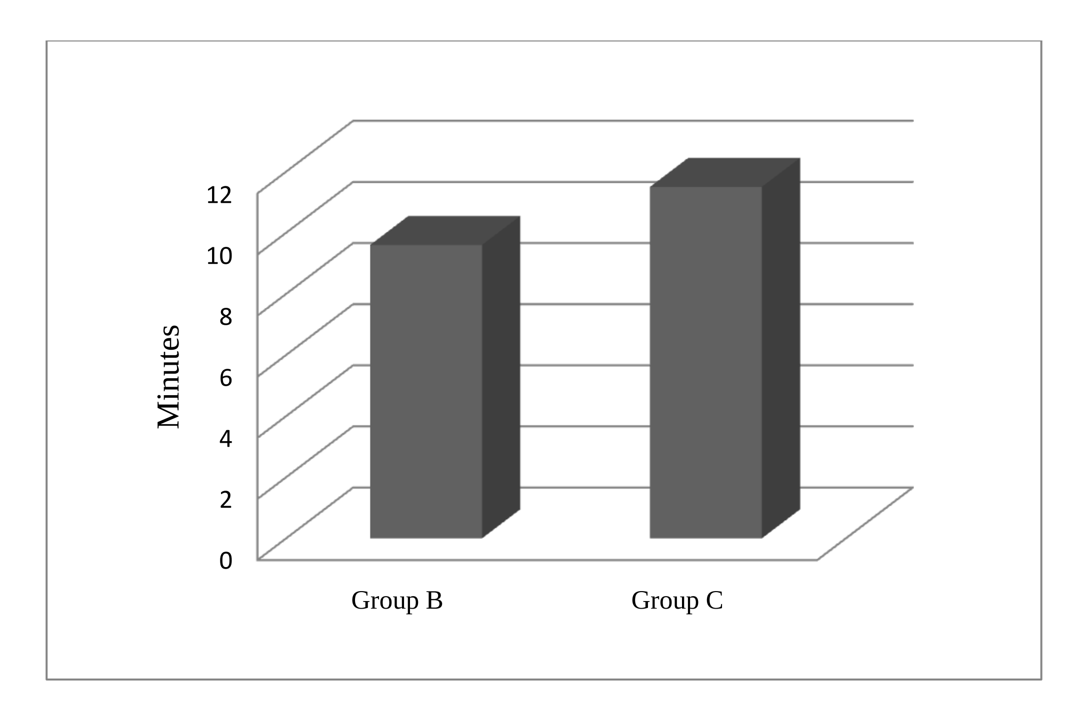


Figure 5.11: Mean waiting time in group B and group C

5.2.4 Extraction Time

Extraction time was recorded for each patient and was calculated in terms of minutes from the beginning of the relatively pain-free extraction until the complete removal of the tooth from its socket. The extraction time for group A ranged from 0.50 to 60.00 minutes with a mean of 6.96 (± 9.42) minutes. For group B, it ranged from 0.25 to 45.00 minutes with a mean of 10.45 (± 11.42) minutes and for group C it ranged from 0.50 to 60.00 minutes with a mean of 9.69 (± 12.34) minutes (Figure 5.12). The differences in the mean extraction time between the three groups were statistically insignificant (P = 0.24).

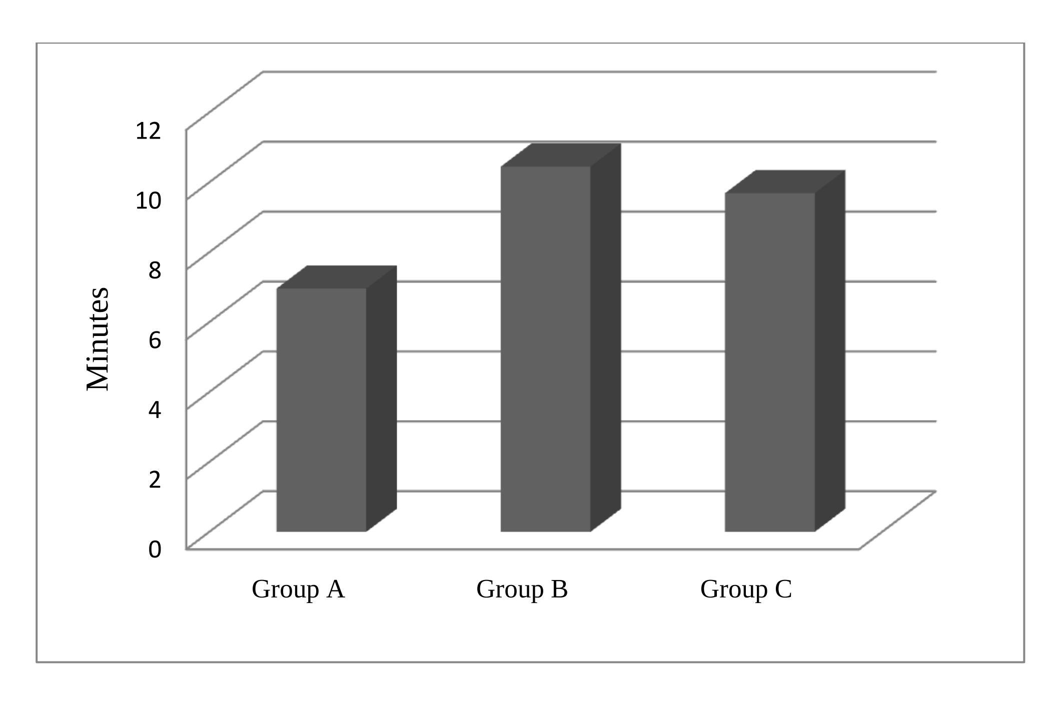


Figure 5.12: Mean extraction time in the three study groups

5.3 Pain Assessment

5.3.1 VAS Score for local anesthetic injection

The scores of the VAS for the local anesthetic injection in group A ranged from 1.00 to 97.00 mm with a mean score of 28.58 (± 24.10) mm. The scores in group B ranged from 0.00 to 42.00 mm with a mean score of 10.24 (± 10.74) mm and the scores in group C ranged from 0.00 to 59.00 mm with a mean score of 12.52 (± 12.57) mm (Figure 5.13). The differences in the mean scores between the three groups were statistically significant ($P < 0.01$). On comparing each two groups separately it was found that the difference between group A and group B was statistically significant ($P < 0.01$) and the difference between group A and group C was also statistically significant ($P < 0.01$). However, the difference between group B and group C was statistically insignificant ($P = 0.60$).

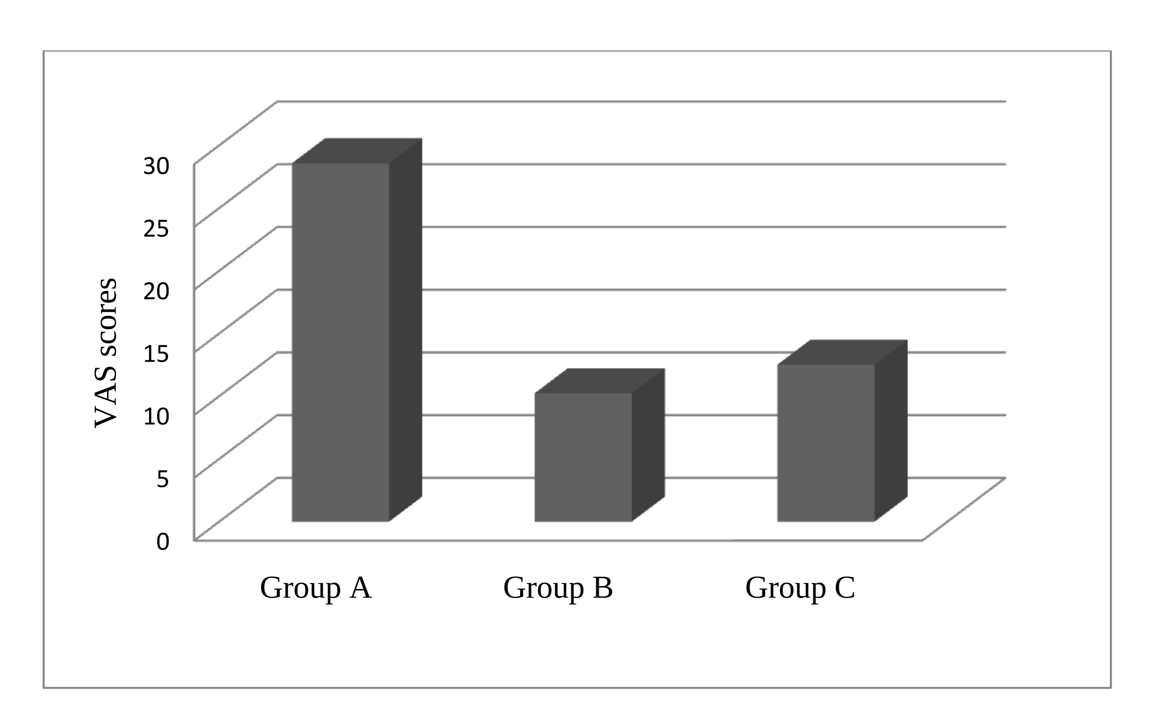


Figure 5.13: Mean VAS scores of local anesthetic injections in the three study groups

5.3.2 VRS1 for local anesthetic injection

In group A, 26 (47.30%) patients reported that the pain of the injections was mild. In group B, 44 (88.00%) patients reported the pain of the injections to be mild and in group C 45 (90.00%) patients reported the pain of the injections to be mild. Moderate pain caused by the injection was reported by 24 (43.60%) patients in group A, 6 (12.00%) patients in group B and 5 (10.00%) patients in group C. Severe pain was reported by only 5 (9.10%) patients in group A (Figure 5.14). The differences in the assessment of local anesthetic injection pain using the VRS1 between the three groups were statistically significant ($P < 0.01$).

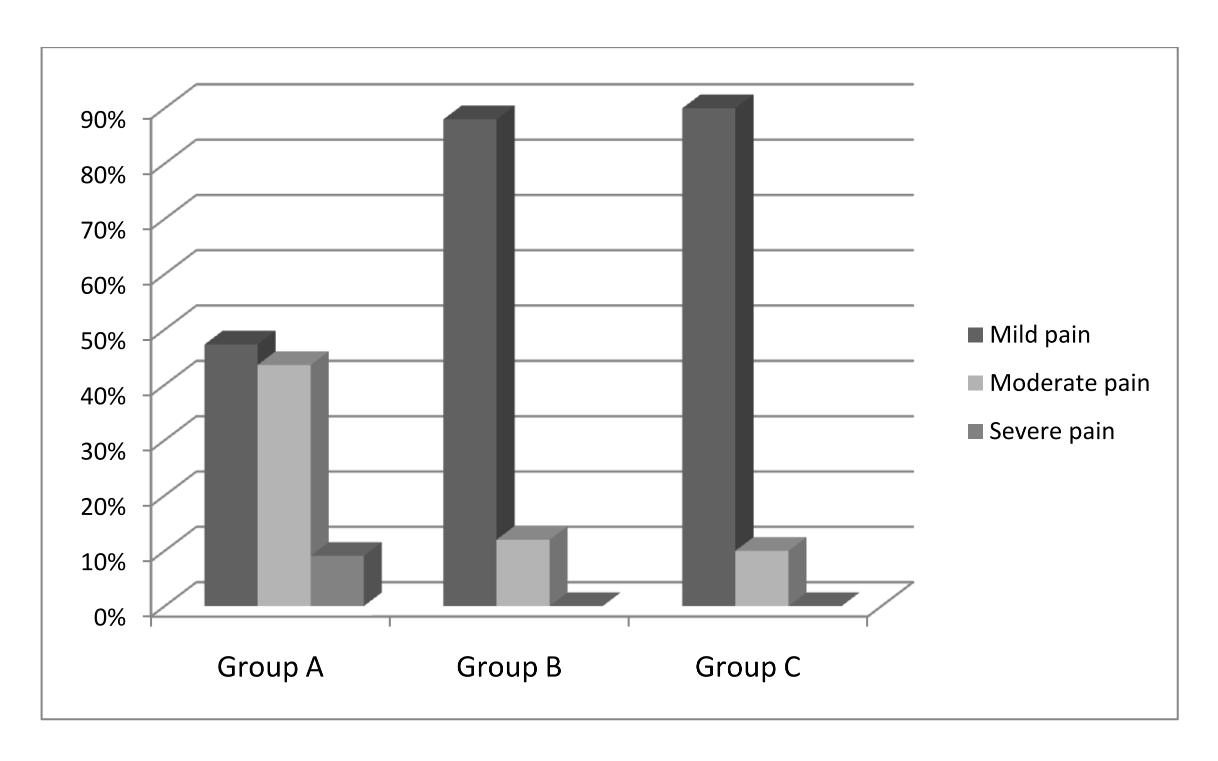


Figure 5.14: Distribution of patients according to injection pain severity on VRS1 in the three study groups

5.3.3 VRS2 for local anesthetic injection

In group A, 35 (63.60%) patients described the pain as less than they expected. In group B, 37 (74.00%) patients described it as less than they expected and in group C, 36 (72.00%) patients described it as less than they expected. In group A, 16 (29.10%) patients described the pain of the injection as they expected. In group B, 10 (20.00%) patients described the pain as they expected and in group C, 14 (28.00%) patients described it as they expected. In group A, 4 (7.30%) patients described the pain as greater than they expected and in group B, 3 (6.00%) patients reported the pain as greater than they expected (Figure 5.15). The differences in the assessment of local anesthetic injection pain by VRS2 between the three groups were statistically insignificant (P = 0.30).

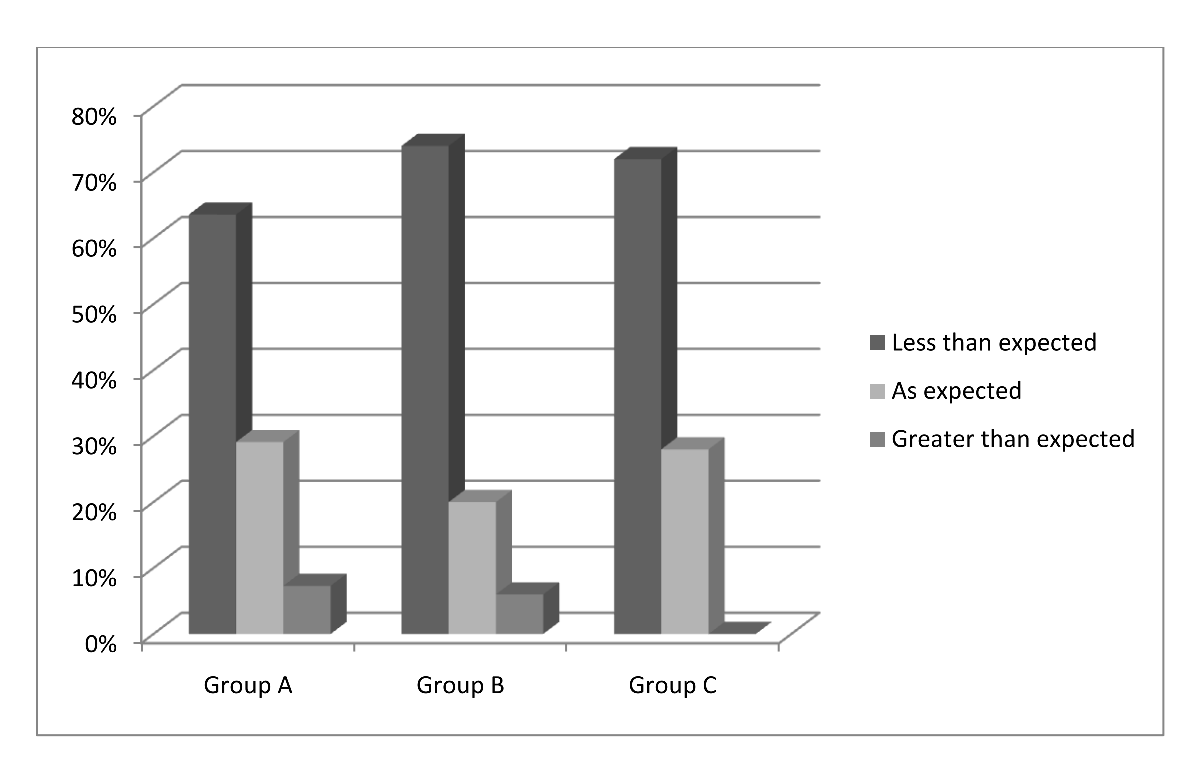


Figure 5.15: Distribution of patients according to injection pain expectation on VRS2 in the three study groups

5.3.4 VAS Score For The Extraction

The scores of the visual analogue scale for the extraction procedure in group A ranged from 0.00 to 89.00 mm with a mean of 19.31 (± 21.25) mm. The scores in group B ranged from 0.00 to 74.00 mm with a mean of 21.30 (± 19.99) mm and for group C they ranged from 1.00 to 75.00 mm with a mean of 21.42 (± 19.66) mm (Figure 5.16). The differences in the VAS scores for the extraction procedure between the three groups were statistically insignificant (*P* =0.84).

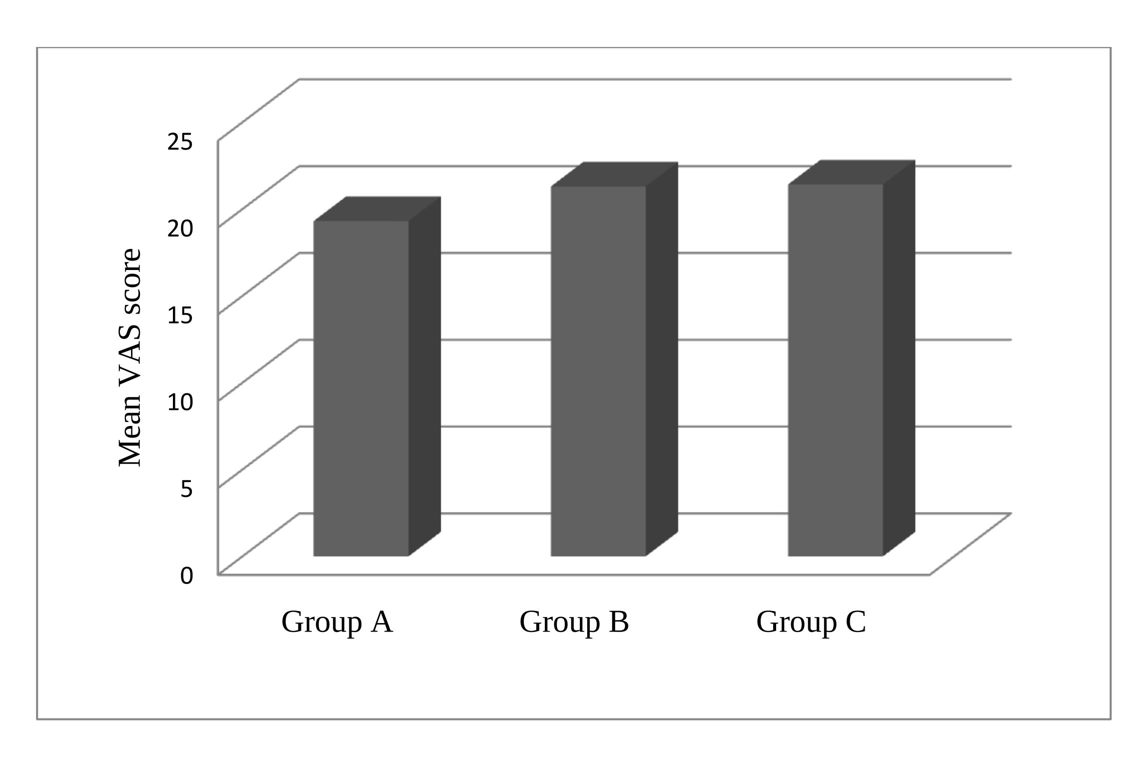


Figure 5.16: Mean VAS scores for the extraction in the three study groups

When the type of injection was ignored and patients were divided into two groups according to gender, the average VAS score of extraction for all females in the study was 24.30 (± 22.03) mm whereas for all males it was 13.16 (± 13.30) mm. The difference in the mean VAS score between the two groups was statistically significant ($P < 0.01$). On the other hand, when the type of injection was ignored and patients were divided into different groups according to the reason of extraction, the differences in the perception of extraction pain between the different groups was statistically insignificant. ($P > 0.05$)

In addition, the correlation between the extraction time and the VAS score of extraction was weak but statistically significant positive correlation (Pearson correlation factor = 0.21, $P = 0.01$).

5.3.5 VRS1 For The Extraction

Mild pain during extraction was reported by 41 (74.50%) patients in group A, 31 (62.00%) patients in group B and 30 (60.00%) patients in group C. Moderate pain was reported by

14 (25.50%) patients in group A, 17 (34.00%) patients in group B and 17 (34.00%) patients in group C. Severe pain was reported by 2 (4.00%) patients in group B and by 3 (6.00%) patients in group C (Figure 5.17). The differences in the severity of the extraction pain using the VRS1 between the three groups were statistically insignificant (*P* = 0.30).

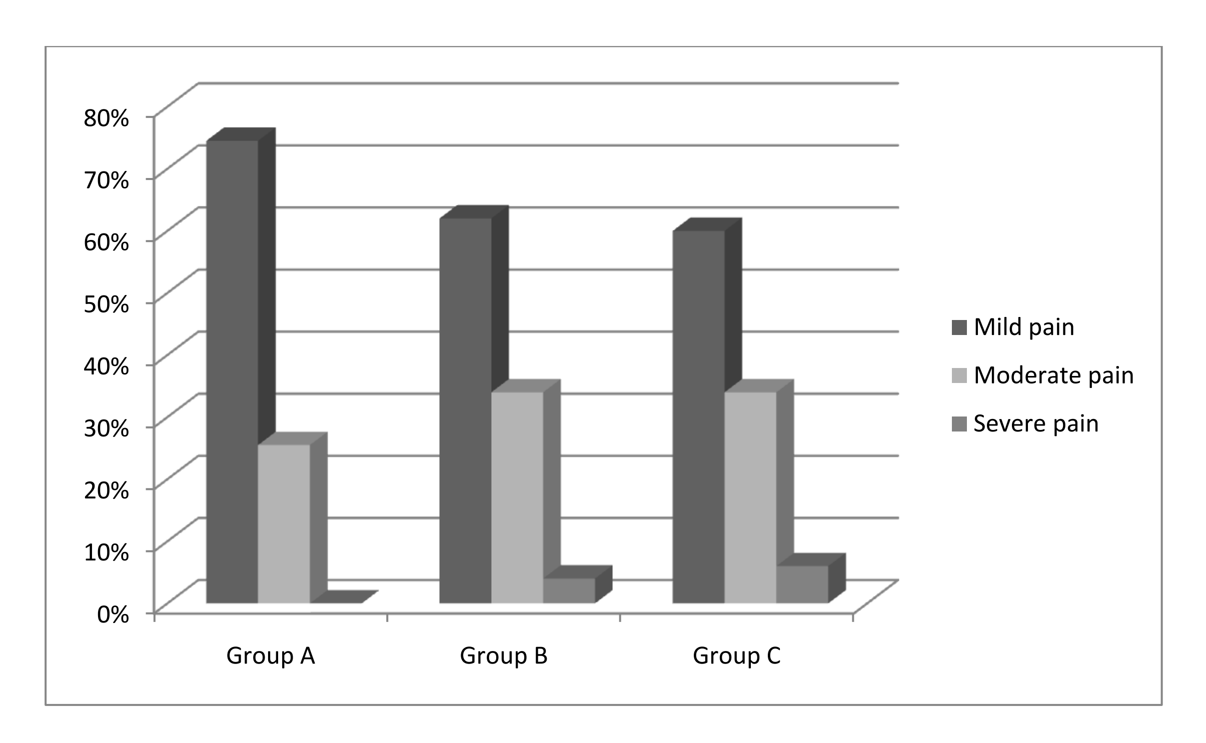


Figure 5.17: Distribution of patients according to extraction pain severity on VRS1 in the three study groups

5.3.6 VRS2 For The Extraction

A total of 41 (74.50%) patients in group A reported the pain as less than they expected. In group B, 33 (66.00%) patients reported the pain as less than they expected and in group C, 35 (70.00%) patients reported the pain as less than expected. In group A, 11 (20.00%) patients described the pain as they expected. In group B, 10 (20.00%) patients described the pain as they expected and in group C, 11 (22.00%) patients described the pain as they expected. In group A, 3 (5.50%) patients described the pain as greater than they expected. In group B, 7 (14.00%) patients described the pain as greater than they expected and in

group C, 4 (8.00%) patients reported the pain as greater than they expected. (Figure 5.18) The differences in the severity of extraction pain using VRS2 between the three groups were statistically insignificant (*P* = 0.64).

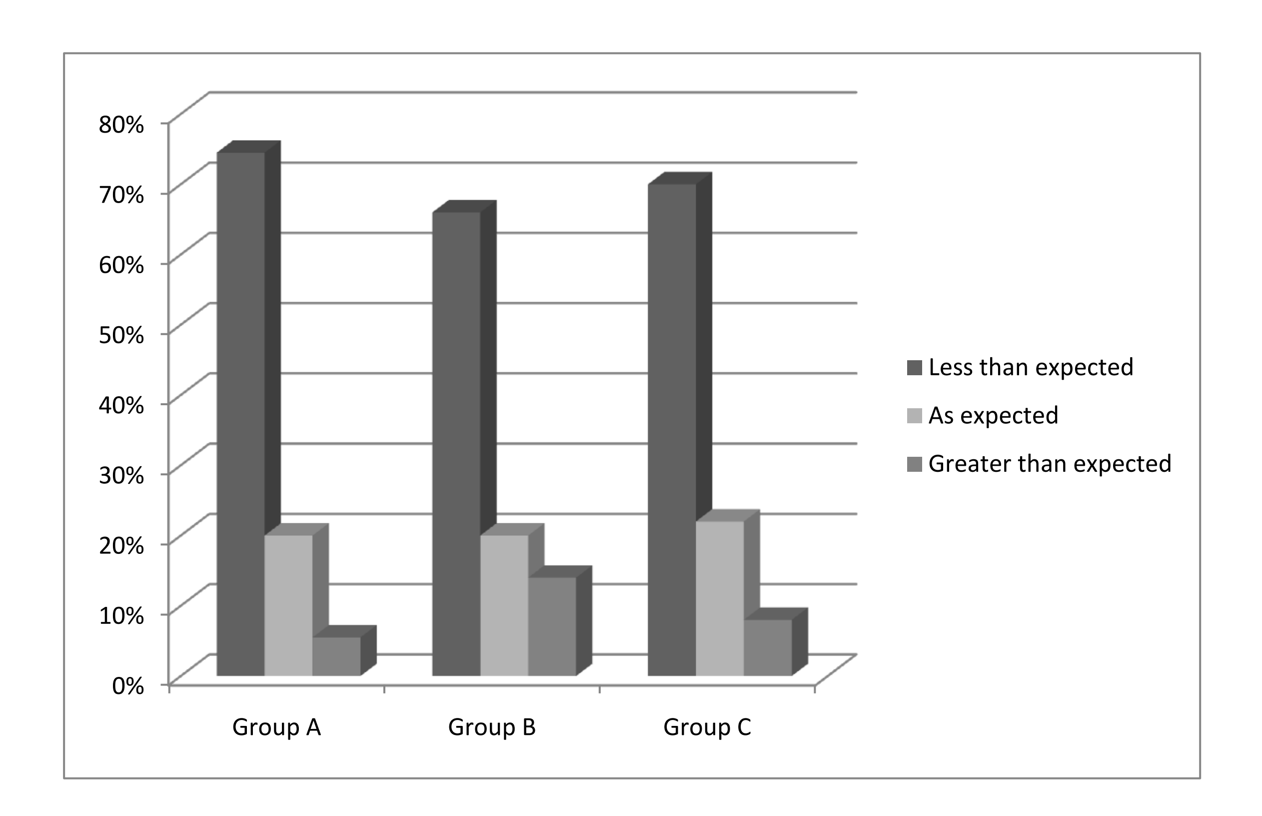


Figure 5.18: Distribution of patients according to extraction pain expectation on VRS2 in the three study groups

Chapter Six: Discussion

The pain of the palatal injection is known to be poorly tolerated by patients (Fan et al, 2009), and although a number of techniques have been advocated to reduce the pain of palatal injections, they have not gained universal acceptance (Duncan et al, 1992) and palatal injections are still a painful experience for patients (Harbert, 1989). Therefore, a recent suggestion of the unnecessity of palatal injections for the extraction of maxillary teeth has gained an increasing interest. The purpose of this study was to evaluate the possibility of extracting maxillary teeth without a palatal injection by comparing patients' perception of pain during the extraction with and without a palatal injection.

In this study, no study power or sample size calculation were done, but it included the extraction of 155 permanent maxillary teeth compared to 76 permanent maxillary teeth in Uckan et al (2006) study, 102 maxillary third molars in Badcock et al (2007) study, 142 permanent maxillary teeth in Fan et al (2009) study, 142 permanent maxillary teeth in Peng et al (2008) study and 200 impacted maxillary third molars in Lima-Junior et al (2009) study. Twenty-three patients had more than one tooth to be extracted and 16 patients acted as their own control in the study. Although Badcock et al (2007) and Fan et al (2009) included only bilateral extraction cases, Uckan et al (2006) included only 23 bilateral extractions and Peng et al (2008) included only 38 patients with bilateral extractions. Bilateral design in this study was not possible due to the presence of three groups, however patients in the three groups were matched according to age, gender, smoking status, tooth to be extracted and the reason of extraction. Patients with bilateral extractions were

included and allocated first either in the experimental group or in the negative control group and for the second extraction were allocated in the positive control group.

6.1 Sample Recruitment Variables

The study population consisted of 131 patients; 47 males and 84 females. The difference in gender distribution between the three groups was statistically insignificant (P = 0.78). Therefore, gender distribution in the groups can be assumed to have no effect on the results of the study and the groups were matched in terms of gender distribution.

The age of patients in the study ranged from 13.00 to 62.00 years with a mean of 30.63 years. The difference in the mean age between the three groups was statistically insignificant (P = 0.82). Thereby, the difference in the mean age between the three groups can be assumed to have no affect on the results and the three groups were matched in terms of patients' age. The age range in this study was similar to that in Badcock et al (2007) study and Fan et al (2009) study; however the mean age in this study was higher. Children below the age of 13 years were not included in this study since it was found that many factors can make evaluating the efficacy of dental anesthesia in children very challenging, including psychologic aspects such as strong anxiety possibly leading to panic, in addition to the difficulty of evaluating pain by some young patients (Sixou and Barbosa, 2008).

Regarding previous experience of extractions in the maxilla, only Uckan et al (2006) considered this variable in the recruitment of their sample with the experience of previous extraction in the maxilla being a condition for inclusion in their study. In the current study, such experience was not a condition for inclusion in the study. However, the differences in the previous extraction experience between the three groups were statistically insignificant (P = 0.98). Thus, this variable can also be assumed to have no effect on the results of the study and the three groups were matched in terms of the previous extraction experience.

The determination of the smoking status of the participants in this study relied on self-reporting rather than measurement of cotinine levels, although it was found that self-report of smoking may underestimate smoking prevalence by up to 4% (Wagenknecht et al, 1992). Participants who reported that at the time of the study they smoked ≥ 5 cigarettes per day were classified as smokers (Horn et al, 2008), whereas any participant who never smoked, formerly smoked and quit more than a month before the study and participants who smoked < 5 cigarettes per day were classified as nonsmokers.

Again, in the current study the difference in the distribution of smokers between the three groups was statistically insignificant (P = 0.79), and should not have affected the results of the study. Regarding the mean amount of smoking, there was no statistically significant difference between the three groups in this study (P = 0.49). Thus, it can safely be assumed that this has not affected the results of the study.

All maxillary permanent teeth from central incisors to third molars were included in this study, which is similar to what was done by Uckan et al (2006) and Fan et al (2009) but different from Badcock et al (2007) who included only maxillary third molars in their study. The difference in the distribution of different teeth between the three groups in this study was statistically insignificant (P = 0.65), and thus it should have not affected the results, and the three groups were matched in terms of tooth distribution.

The reasons for extraction of teeth in this study varied. They included caries, orthodontic reasons, cheek biting, pericoronitis, prosthodontics reasons, endodontic failure, nonfunctional third molars and remaining roots. Differences in the distribution of reasons of extraction in the three groups in the current study were statistically insignificant (P = 0.61), and should have not affected the results of the study.

Teeth with periodontal disease or any mobility were excluded from this study because the decreased bony support around these teeth may facilitate the diffusion of buccally

administered local anesthesia to the palatal side, which would adversely affect the accuracy of the results of the study. Similarly, symptomatic teeth that needed emergency extraction due to acute irreversible pulpitis were excluded from the study since it is believed that the effectiveness of local anesthetics decreases in these cases. In fact, it was found that teeth with irreversible pulpitis demonstrated an eightfold higher rate of local anesthetic failure compared to teeth with normal vital pulps (Rosenberg et al, 2007). In addition, an appropriate nerve block injection as far back as possible along the innervation pathway of the hypersensitive tooth is often needed for anesthetizing such teeth (Budenz, 2003), whereas the study protocol was to give only supraperiosteal injections in the buccal sulcus. Furthermore, teeth with any sign of symptomatic periapical infection or surrounding inflammation were also excluded because during inflammation polymodal nociceptors are sensitized and their excitation threshold drops that even light normally innocuous stimuli will activate them to cause pain.

6.2 Procedure Variables

Intraoperative complications that were encountered during the extraction procedure included fracture of the tooth, whether fracture of the crown or of the root, fracture of maxillary tuberosity, trauma to soft tissues, luxation of an adjacent tooth, intolerable pain and oroantral communication. Supplemental anesthesia according to the protocol of the study was administered to patients with intolerable pain. The difference in the occurrence of intraoperative complications between the three groups was statistically insignificant (P = 0.72). Therefore, the difference in the occurrence of different intraoperative complications between the three groups did not affect the results of the study.

The amount of buccally administered local anesthesia was recorded for each patient as the number of cartridges administered until complete removal of the tooth. The number of cartridges was used as the measuring unit because it is simple, easy and standardized.

Fractions of cartridges were approximated to the nearest 1/4 of cartridge as judged by the researcher. Since one researcher did all the procedures and measurements, any errors in measurement can be ignored since they would apply equally to the three groups of the study.

The mean amount of buccally administered local anesthesia was significantly different between the groups, being lowest in group A ($P < 0.01$). The mean amounts given to groups B and C were comparable, but higher than that given to group A. This clearly demonstrates that if the palatal injection is to be avoided, a larger amount of the buccally administered local anesthesia will be needed. This is most likely because the concentration of the local anesthetic agent decreases as it diffuses through a larger volume of tissues. Thus, if a small amount is given buccally, it will not reach the palatal side at a high enough concentration to achieve adequate anesthesia, assuming that it can diffuse to reach the palatal side. To compensate for this drop in concentration, a larger volume of anesthetic solution is needed.

It is also possible that these differences was due to patients not being blinded to the administered anesthesia which might made them more sensitive to these differences. Nevertheless, this might be acknowledged as a possible criticism. Badcock et al (2007) explained that by the fact that a number of patients might require ‘top up’ local anesthetic and they stated that whether a potential small increase in the frequency of the need of additional anesthesia is preferred over the administration of a palatal injection is to be determined by the individual operator.

The assumption that an increase in dosage of local anesthetic also increases its efficacy is not entirely supported by the literature. In fact, it was found that once the effective volume of local anesthetic is attained, no further benefit can be achieved through a further increase in anesthetic dosage, and in the case of 2% lidocaine with adrenaline, this effective volume

has been reported to be 2 ml of anesthetic solution (Tortamano et al, 2009). Therefore, all patients were given only 1 cartridge before beginning the extraction procedure and additional anesthesia was given only when it was judged that it was needed to complete the procedure. In the current study, larger amounts of buccally administered local anesthesia were needed in groups B and C in an attempt to provide effective volume and concentration at the palatal side.

The protocol of local anesthetic administration followed in the current study allowed adequate waiting time and amount of local anesthesia in groups B and C for the anesthetic agent to diffuse to the palatal side, so that if palatal anesthesia was not achieved at the end of the procedure, it would be the result of inability of the anesthetic solution to diffuse through thick tissues and anatomical barriers rather than inadequate amount or waiting time. Other studies on the same subject followed similar, but slightly different protocols of giving an initial amount of local anesthesia, waiting a specific period of time and increasing the amount of anesthetic solution if needed (Fan et al, 2009, Uckan et al, 2006, Badcock et al, 2007).

In the current study, waiting time was recorded only for groups B and C and was not recorded for group A where palatal injections were given, which obviated the need to wait for the diffusion of the anesthetic agent. Even though the mean waiting time in group C was longer than that in group B, the difference between the two groups was statistically insignificant ($P = 0.06$). This suggests that the diffusion characteristics of articaine and lidocaine are not significantly different. Moreover, the waiting time in the articaine group ranged from 5.00 to 30.00 minutes with a mean waiting time of 11.50 (± 5.91) minutes, which is similar to the findings of Lima-Junior et al (2009) who reported that higher diffusion of articaine was obtained after a waiting period of 10 minutes, but different from

the findings of Uckan et al (2006) where all teeth in the articaine group were extracted after a waiting time of 5.00 minutes.

Extraction time was recorded for each patient and was calculated in minutes from the relatively pain-free application of the elevator or the forceps until the complete removal of the tooth from its socket. The differences in the mean extraction time between the three groups were statistically insignificant (P = 0.24). This suggests that extraction difficulty was comparable between the three groups, which give more strength to the findings of the study.

6.3 Study Results

The previous belief of the necessity of a palatal injection to attain adequate anesthesia for the extraction of permanent maxillary teeth is no longer absolute. One of the aims of this study was to compare the patient's perception of pain of the palatal injection with that of the buccal injection, in addition to comparing their perception of pain during the extraction of permanent maxillary teeth without a palatal injection with that with a palatal injection. The study also aimed to compare the efficacy of articaine 4% with 12 μg/ml epinephrine with that of lidocaine 2% with 15 μg/ml epinephrine when they are administered as a buccal injection only for the extraction of permanent maxillary teeth.

The perception of the pain of the injection and the pain perception during the extraction were assessed using one visual analogue scale and two verbal response scales. As suggested by Briggs and Closs (1999) the VAS scores were obtained first and the VRS scores second to minimize bias caused by patients trying to match the word chosen with a mark on the VAS. Other studies used measurements from a pulp tester (Mikesell et al, 2005) or heart rate (Bigby et al, 2006). Measuring the anesthetic effect with a pulp tester or heart rate is likely to be less subjective than asking patients to rate their level of pain on the VAS. However, the main advantage of the VAS and VRS is that they directly measures the

level of discomfort perceived by the patient, and this might be easier to translate to clinical practice, in comparison to pulp test or heart rate measurements.

The VAS and VRS are among the most commonly used scales for pain assessment in research and clinical practice. High correlations have been shown between the VAS and the VRS. In addition, both scales are considered to have good reliability and validity when self report is used as the method of data collection. Moreover, they are easy to use and place minimal demands on almost all patients (Briggs and Closs, 1999, Fan et al, 2009).

Since pain perception is complex and multidimensional, and the VAS and VRS are unidimensional and have their own limitations, the use of more than one scale for pain assessment was recommended by many authors. While the VAS is more sensitive and allows the use of parametric statistical methods, the VRSs are easier to administer, especially for elderly people and allow the use of nonparametric statistical methods (Briggs and Closs, 1999, Oliveira et al, 2004). Therefore, three different scales were used in the current study to increase the reliability of the results. Other studies on the same subject also used a combination of VAS and VRS to assess the patients' perception of pain during the anesthetic injections and the extractions (Uckan et al, 2006, Fan et al, 2009, Badcock et al, 2007).

The mean VAS score for the injections was comparable between groups B and C, in which it was significantly lower than that of group A ($P < 0.01$). This is not surprising since patients in group A received palatal injections, which are known to be significantly more painful than the buccal injections only given to patients in group B and C. This difference in pain perception between buccal alone and buccal with palatal injections is similar to the findings of Fan et al (2009). In addition, the type of anesthetic solution made no difference in injection pain perception as can be seen by comparing the results of group B with those

of group C. This is in agreement with the findings of Kanaa et al (2006), Robertson et al (2007) and Evans et al (2008).

The results of the VAS regarding the injection pain were mirrored by the results of VRS1, where the vast majority of patients in groups B and C rated the injection pain as mild, a few of them rated it as moderate, and none of them rated it as severe. On the other hand, only half of the patients in group A rated the injection pain as mild, many of them rated it as moderate and some of them rated it as severe.

When the results of VRS2 are considered, it becomes evident that the vast majority of patients expect the injections to be more painful than they actually are. Moreover, it seems that patients in general expect the palatal injection to be more painful than buccal injections. This is probably why there was no significant difference between the three groups in terms of their expectation for the injection pain regardless of the injection technique or solution. It is likely that previous experience prepares patients to what to expect from the injections as the majority of patients in this study had previous extractions and hence previous injections in the maxilla.

When the VAS scores for the extraction itself are considered, the results of the current study show no significant difference between the three groups. This not only supports the emerging concept that palatal injections are not necessary for painless extraction of maxillary teeth, but also that articaine is not different from lidocaine in this regard. The same can be concluded by examining the VRS1 scores for the pain of the extraction, which were comparable between the three groups. The vast majority of patients in the three groups rated the extraction pain as either mild or moderate, with only a very small minority in groups B and C rating their pain as severe. These differences may be explained by different pain thresholds between different patients and the intense pressure felt by some patients during the extraction, which may be difficult to differentiate from pain.

Regarding patients' expectation of extraction pain, the scores of the three groups on VRS2 suggest that neither the palatal injection, nor the type of anesthetic solution made any difference in extraction pain perception in comparison to patients' expectations, as there was no significant difference between the three groups. The fact that the majority of patients in the three groups found the pain less than they expected is difficult to explain in view of the fact that the majority of them had maxillary extractions before they participated in this study. It is possible that the fear of tooth extraction exaggerates patients' expectations before the actual extraction.

The results of the VAS and the VRS for the extraction suggest that the extraction of maxillary teeth without the administration of a palatal injection is possible with the buccal administration of either articaine or lidocaine. Fan et al (2009) explained that by the relatively thin and porous bone of the buccal maxilla which facilitates the diffusion of any local anesthetic. The claim that articaine diffuses more readily in tissues than other local anesthetics (including lidocaine) was not demonstrated in this study.

This study was designed so that a palatal injection would be given to any patient in group B or C if the extraction was still painful after a waiting time of 30 minutes after the first local anesthetic injection. Palatal injections were needed in 2 patients in group B and 3 patients in group C after 30 minutes of the first buccal injection and 15 minutes of the second buccal injection since these patients still experienced pain on the commencement of extraction. This makes the success rate of maxillary tooth extraction without palatal injection 94% for articaine and 96% for lidocaine. The slightly higher success rate with lidocaine can be explained by the higher epinephrine concentration in the lidocaine anesthetic solution (0.015mg/ml) as compared to articaine anesthetic solution (0.012 mg/ml). This is in agreement with Lima-Junior et al (2009) results of higher success rate with higher vasoconstrictor concentrations. However, the success rates of this study are

different from those of Fan et al (2009) and Badcock et al (2007) who reported a 100% success rate.

When the type of injection was ignored and the perception of pain was compared according to gender, the perception of extraction pain was significantly higher in females than males ($P < 0.01$), which can be explained by the belief of the lower threshold of pain in females (Vallerand and Polomano, 2000). In addition, it was also found that the perception of extraction pain increases with increased time of extraction, which is consistent to the findings of Badcock et al (2007). On the other hand, when the type of injection was ignored, it was found that the reason of the extraction of the tooth did not affect the perception of extraction pain, which is different from the findings of Fan et al (2009).

The findings of this study in general are in agreement with those of Uckan et al (2006), Badcock et al (2007), Peng et al (2008), Lima-Junior et al (2009) and Fan et al (2009), which collectively suggest that painless extraction of maxillary permanent teeth without a palatal injection is possible.

Chapter Seven: Conclusion

Based on the results of this study, the following can be concluded:

1. The palatal injection is significantly more painful than the buccal injection.

2. The extraction of permanent maxillary teeth without a palatal injection is not associated with more significant pain than their extraction with a palatal injection.

3. The palatal injection is associated with significant pain and it does not appear to significantly reduce extraction pain.

4. The extraction of permanent maxillary teeth seems to be possible in the vast majority of cases without the administration of palatal injection.

5. There is no difference between articaine 4% and lidocaine 2% in their ability to produce adequate palatal anesthesia when they are administered in the maxillary buccal vestibule.

6. A waiting period of at least 5 minutes is probably needed after the administration of local anesthesia buccally to allow for the diffusion of the local anesthetic agent to produce adequate palatal anaesthesia.

Chapter Eight: References

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Appendix (A)

موافقة على الاشتراك في بحث طبي

نطلب منك المشاركة في بحث طبي دراسي " مقارنة بين ادراك المرضى الحسّي للألم بعد قلع الأسنان العلوية بدون حقنة سقف الحلق مع قلعها مع حقنة سقف الحلق " للطـــــالبة رشا قاسم الرحاحلة تحت إشراف الدكتور ينال محمد نصير.

لكن قبل أن توافق , يجب على الباحث إخبارك عن:

1- أهداف وإجراءات ومدة هذا البحث

2- أي إجراءات تجريبية

3- أي إجراءات بديلة التي ربما يكون لها منافع

4- كيفية المحافظة على سرية معلوماتك الشخصية

ويجب على الباحث إخبارك أيضا عن:

1- ظروف معينة يضطر منها الباحث وقف مشاركتك في البحث

2- ماذا يحدث إذا قررت أنت أن تنهي مشاركتك في البحث

3- عدد المشتركيين في هذا البحث الطبي الدراسي

إذا وافقت على المشاركة في هذا البحث ,يجب أن تعطى نسخة من هذه الوثيقة لتوقيعها.

في حال وجود أي أسئلة لديك يمكن الأتصال على رقم هاتف 0795519018

مساهمتك في هذا البحث مساهمة خيارية وتطوعية .إذا قررت أن لا تشارك أو إذا شاركت في البحث ثم قررت أن توقف مساهمتك لن يتخذ أي إجراء ضدك ولن تخسر أي فوائد.

توقيعك على هذه الوثيقة يعني انه قد تم شرح هذا البحث الطبي الدراسي لك شفويا مع كل المعلومات المذكورة أعلاه وانك قد قررت المشاركة التطوعية في هذه الدراسة.

توقيع المشارك في البحث التاريخ

توقيع شاهد التاريخ

Appendix (B)

Group: …………………………

Biographic data:

Name :…………………………………… Gender:……. DOB:………… Marital status:………………………… address:…………………………………. Contact number:…………………….. file number:…………………………… Occupation:…………………………..

Medical history:

Anemia	
Bleeding disorders	
Cardiorespiratory disease	
Drug treatment & allergy	
Endocrine disease	
Fits & faints	
Gastrointestinal disorders	
Hospital admissions	
Infections	
Jaundice & liver disease	
Kidney disease	
Likelihood of pregnancy	
Others	

Dental history:

☐ Previous extractions : ☐ maxilla ☐ mandible

☐ Others …………………………..

Oral hygiene habits:

☐ Brushing ………/day ☐ flossing……../day ☐ miswak ……/day

Social history

☐ Smoking ……../day ☐ alcohol

Tooth/teeth that needs to be extracted:

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

Reason of extraction:

☐ Caries ☐ Prosthodontics ☐ Remaining root

☐ Orthodontics ☐ Cheek Biting ☐ Pericorontis

☐ endodontic failure ☐ other reason ……………………….

Waiting Time: ……………………….

Number of received Cartridges buccally: …………………. Palatally: ……………..

Intraoperative complications:

☐ None ☐ trauma to soft tissue ☐ damage to adjacent tooth

☐ Pain ☐ Bleeding ☐ fracture

Others ……………………………

* Time of extraction :……………….

* VAS:

Injection

No pain at all Unbearable Pain

extraction

* VRS:

Injection: ☐ Mild pain ☐ moderate pain ☐ severe pain

☐ Less than expected ☐ as expected ☐ greater than expected

Extraction: ☐ Mild pain ☐ moderate pain ☐ severe pain

☐ Less than expected ☐ as expected ☐ greater than expected

إدراك المرضى الحسي للألم أثناء حقنة الأرتكين أو الليدوكين و قلع الأسنان الدائمة العلوية. مقارنة بين الحقنة الفحوية و الحقنة الفحوية مع حقنة سقف الحلق

إعداد: رشا قاسم الرحاحلة

الملخص

تشمل الطريقة التقليدية لإعطاء مخدر موضعي قبل قلع أي سن علوي كما تُعلّم لأطباء الأسنان إعطاء المريض حقنة فحوية و حقنة في سقف الحلق، و ذلك لتخدير الأنسجة العظمية و اللحمية بهدف قلع غير مؤلم. تُعرف حقنة سقف الحلق بأنها مؤلمة، و قد ظهرت في السنوات الأخيرة دراسات تشير إلى عدم ضرورة حقنة سقف الحلق لقلع الأسنان العلوية. الهدف من هذه الدراسة تقييم الحاجة إلى حقنة سقف الحلق عند قلع الأسنان العلوية و ذلك بمقارنة الإدراك الحسي للألم عند المرضى خلال قلع الأسنان العلوية مع حقنة سقف الحلق و قلعها بدون حقنة سقف الحلق، و تقييم قدرة الأرتيكين على تخدير أنسجة سقف الحلق عند إعطاء حقنة فحوية فقط و مقارنته مع الليدوكين.

لقد تم قلع مئة وواحد وخمسين سناً من مئة و واحد و ثلاثين مريضاً ممن اشتملت عليهم الدراسة. و قد تم توزيعهم على ثلاث مجموعات. التخدير الموضعي للمجموعة الأولى كان بإعطائهم ليدوكين كحقنة فحوية و حقنة في سقف الحلق، و للمجموعة الثانية كان بإعطائهم حقنة ليدوكين فحوية فقط، أما للمجموعة الثالثة فكان بإعطائهم حقنة أرتيكين فحوية فقط. و قد تم تقييم الإدراك الحسي للألم عند المرضى بعد إعطاء الحقنة و بعد القلع بإستخدام مقياس الألم البصري و مقياس الألم اللفظي.

لقد تبين أن الإدراك الحسي للألم بعد الحقنة الفحوية يختلف إحصائيا عن الإدراك الحسي للألم بعد حقنة سقف الحلق، بينما الإدراك الحسي للألم خلال القلع لم يختلف إحصائيا مع وجود حقنة سقف الحلق أو بدونها وفي النهاية أظهرت نتائج الدراسة أن قلع الأسنان العلوية بدون حقنة سقف الحلق ممكن في معظم الحالات وأنه لافرق بين الليدوكين والأرتيكين عند استخدامهما كحقنة فحوية لقلع الأسنان العلوية.